The Journal of Thoracic Surgery

VOL. 28 DECEMBER, 1954 No. 6

Original Communications

THE SURGICAL CORRECTION OF MITRAL INSUFFICIENCY BY THE USE OF PERICARDIAL GRAFTS

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MITRAL regurgitation or insufficiency has long been recognized as a common accompaniment of mitral stenosis. Indeed, until it became possible to examine the mitral valve digitally during life, it was generally believed by internists and pathologists alike that mitral insufficiency coexisted in every valve with significant mitral stenosis. The inaccuracy of this concept has been pointed out in previous writings[1-3] and is now generally recognized. In 984 consecutive patients who have been operated upon by the authors in two Philadelphia hospitals, for mitral valve disease, there were found to be 635 (64.5 per cent) cases with pure mitral stenosis, 56 (5.7 per cent) with pure mitral insufficiency, 176 (17.9 per cent) with predominant mitral stenosis and insignificant mitral insufficiency, 69 (7.0 per cent) in which both the mitral stenosis and insufficiency were of approximately equal physiologic significance and 48 (4.9 per cent) cases with significant insufficiency and insignificant stenosis.

In our total experience we have recognized 441 instances of mitral regurgitation, great or small, during intracardiac surgical exploration. While the majority of these leaks were small and of negligible clinical significance, it soon became evident that well-developed mitral regurgitation is a much more serious

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This work was supported by The Mary Bailey Foundation for Heart and Great Vessel Research, by The National Council of Brith Sholom Women, and by The U. S. Public Health Grant #H-394.

Read at the Thirty-fourth Annual Meeting of The American Association for Thoracic Surgery at Montreal, Quebec, May 3-5, 1954.

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lesion than mitral stenosis. Indeed, we feel that clinically it is at least equal to marked aortic insufficiency in its effect upon the integrity of the circulatory system.

Strangely, the profession has been extremely slow to recognize this. Usually, mitral insufficiency has been considered relatively benign. However, White[4] clearly points out its seriousness in his textbook. Probably a part of this lack of understanding has been based upon the presumption that most apical systolic murmurs are indicative of mitral incompetence. Actually, significant mitral regurgitation may be present or absent regardless of a systolic murmur. Furthermore, a small leak may cause as loud a murmur as a large one. Undoubtedly, MacKenzie's teaching[5] that the existence of a specific type of valve lesion was unimportant in comparison with the health of the cardiac muscle has, in the past, seemed to lessen the importance of differentiating mitral stenosis from regurgitation. This concept probably had practical clinical advantages at a period when surgical repair of deformed valves was unthinkable. Now, however, the time has come to discard outmoded and dogmatic *pronunciamentos*.

Actually, the effects of a major mitral regurgitation upon the circulation are most profound and serious. The pulmonary vascular bed becomes congested and hypertensive, much as in severe mitral stenosis. Simultaneously, the left atrium becomes dilated, often to a greater degree than in mitral stenosis, and the right ventricle undergoes comparable dilatation and hypertrophy and eventually fails. The usual phenomena associated with such right-sided heart failure are then observed (hepatomegaly, ascites, systemic venous distention, dependent edema, pleural effusion, etc.).

Unlike the condition encountered in mitral stenosis in which the left ventricle remains small and relatively healthy, protected by the mitral obstruction from overfilling and hence from overwork, in significant mitral insufficiency the left ventricle undergoes great dilatation and hypertrophy. This is necessary for the maintenance of even a minimally adequate aortic output since a portion of each ventricular stroke volume is "wasted" by escape through the incompetent mitral valve into the dilated left atrium. Obviously, this is less prominent with smaller leaks, and is insignificant in minimal ones even though a persistent systolic murmur may be heard. If the total left ventricular output falls off for any reason, a larger proportion is lost through the incompetent mitral valve and the aortic output is disproportionately reduced. Hence, depression from surgery or blood loss is very poorly tolerated in these cases (Fig. 1, *A* and *B*).

The severity of the clinical course in mitral insufficiency (and severity of prognosis for duration of life) is directly proportional to the size of the leak. Undoubtedly, it is inversely proportional to the vigor and tone of the left ventricular myocardium. Presumably, as will be indicated later on, the type of the lesion causing the incompetence is also of primary importance since a coexisting stenotic element may tend gradually to reduce the magnitude of the leak, while insufficiency due to annular dilatation tends progressively to become worse.

Mitral regurgitation may be classified either physiologically according to the size of the leak, or pathologically according to the type and extent of the anatomical abnormalities which exist.

PHYSIOLOGIC CLASSIFICATION

Physiologically the insufficiency may be divided simply into plus 1, 2, 3, or 4 grades of severity according to the magnitude of the leak per beat. This may be reflected by the clinical features of disability, increase in the cardiac size, "paradoxical" pulsations of the left atrium upon fluoroscopy, and a specific pattern on the "wedge" tracing (capillary pressure curve) obtained by cardiac catheterization. There is great difficulty, however, in drawing sound conclusions even from this last type of investigation. The frequency of atrial fibrillation (present in 71.3 per cent of those of our patients who were definitively operated upon for this lesion) seriously limits the conclusions which can be drawn from such pressure curves.

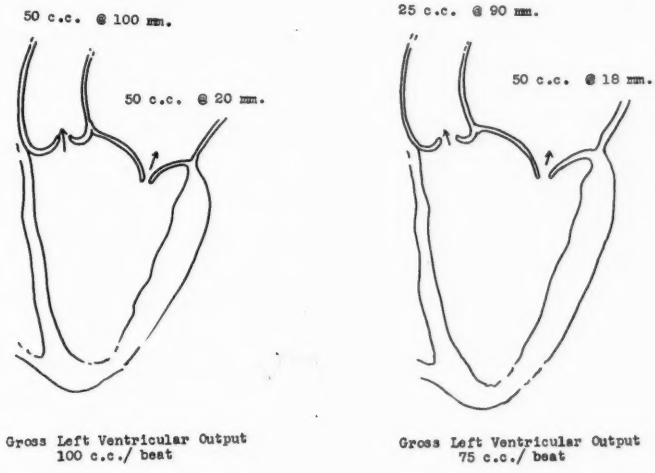


Fig. 1.—*A*, In severe mitral insufficiency, a significant portion of the left ventricular output is wasted by reflux into the low pressure left atrium. *B*, If the left ventricular tone becomes diminished, its total output falls off, but a larger proportion of this goes back into the left atrium, causing a much greater reduction in the effective aortic output.

Ultimately, no doubt, physiologists will develop a method for accurate measurement of the volume of blood lost from the left ventricle per heartbeat. Until that time, there is no better guide to the magnitude of the insufficiency than digital estimation, by intra-atrial examination, of the size of the regurgitant jet It is realized, of course, that this varies with the level of the systolic blood pressure and the vigor of the ventricular contractions and that the velocity and cross-sectional area of the column or "bolus" of regurgitated blood may produce a varying tactile sensation. However, with experience and the application of common sense a surgeon can come to a reasonably reliable conclusion

as to the magnitude of the insufficiency in any given case. Certainly he will be able to detect any significant alterations in the regurgitation which may occur as a result of surgical manipulations.

In order to obtain some reasonable consistency in estimates by various operators, we have attempted to achieve a rough quantitation of the magnitude of the leak. The tactile sensation associated with palpation of a regurgitant jet of blood may be experienced vicariously by ejecting measured amounts of water from a syringe against the tip of a finger immersed in a basin of water. By using syringes with different sizes of opening, an appreciation of the differences in tactile perception of a large or small caliber jet may be attained. It is realized that there are gross elements of inaccuracy in this evaluation, and that only a considerable experience will render a surgeon reliable in his estimates. At the present time, however, there is no better method of evaluating the size of the valvular leak.

We consider as 1-plus an insufficiency in which there is a very small regurgitant jet, varying from the very least perceptible to a slightly more prominent one. Probably such leaks amount to no more than 5 c.c. per heartbeat.

Two-plus regurgitations are somewhat larger, perhaps amounting to 10 c.c. leaks per heartbeat.

Three-plus regurgitations are appreciably larger, probably ranging from 10 to 30 c.c. per beat.

A 4-plus insufficiency is a very large one. It may well amount to over 100 c.c. of regurgitation with each cardiac systole.

In Table I, we have classified 349 cases of mitral regurgitation in which a digital estimation of the leak was recorded.

TABLE I. PHYSIOLOGIC CLASSIFICATION IN 349 PATIENTS WITH MITRAL INSUFFICIENCY, IN AUTHOR'S SERIES

GRADE	NUMBER	PER CENT
I (1 to 5 c.c.)	99	28.3
II (5 to 10 c.c.)	77	22.1
III (10 to 30 c.c.)	137	39.3
IV (30 c.c. and above)	36	10.3
Total	349	100

CLASSIFICATION ACCORDING TO PATHOLOGY

Briefly stated, mitral insufficiency may be of congenital, traumatic, functional, or organic origin (Table II).

TABLE II

CLASSIFICATION OF TYPES OF VALVE IN MITRAL INSUFFICIENCY (PATHOLOGIC)	NUMBER OF CASES	PERCENTAGE
Functional insufficiency	58	13.7
Organic or mechanical insufficiency	158	37.3
Traumatic insufficiency		
Insufficiency created	153	36.1
Insufficiency increased	47	11.0
Congenital insufficiency (Isolated valvular lesion)	8	1.9
Total	424	100.0

Congenital Mitral Insufficiency.—While, no doubt, there may be many different congenital malformations of the mitral valve which can produce insufficiency, we have encountered only two types. Three patients have had one or more perforations of one or both valve leaflets. In five instances mitral insufficiency was recognized during digital examination of the mitral valve in patients with atrial septal defects of the persistent ostium primum type. In two instances it was proved subsequently to be due to a congenital cleft in the septal leaflet of the mitral valve (Fig. 2, *A*, *B*, and *C*).

Traumatic Insufficiency.—Occasionally, mitral regurgitation may be produced by a stab or gunshot wound of the thorax. Adam[6] has reported an instance of traumatic mitral insufficiency due to bullet injury of the valve leaflets. However, this must be an extremely rare occurrence clinically since patients rarely survive such cardiac injury.

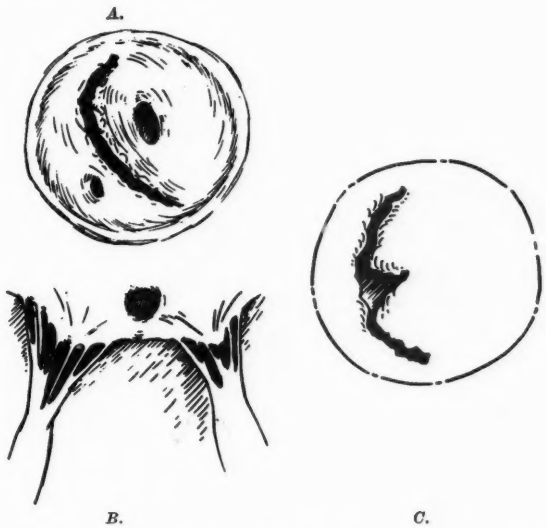


Fig. 2.—*A*, Congenital mitral insufficiency due to perforation of a leaflet. *B*, Note accurate coaptation of valve lips. *C*, Congenital mitral insufficiency due to primitive cleft in septal leaflet. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

A much more common type of traumatic injury now that surgery for mitral stenosis has become a frequent undertaking is the inadvertent creation of regurgitation during attempts at surgical mobilization of the fused valve cusps. This has been observed in 24.8 per cent of a series of 635 consecutive patients with pure mitral stenosis and 26.7 per cent of 176 cases with mitral stenosis associated with insignificant (1-plus) regurgitation. It was of no clinical consequence in the vast majority of the cases, merely representing a recommended extension of the commissural separation as far as technically feasible up to the point of the first detectable beginning of regurgitation. In a few instances, however, the produced regurgitation was serious as indicated by an operative mortality of 8.8 per cent in these cases in contrast to a 7.9 per cent mortality in those in whom no insufficiency was produced.

This kind of traumatic insufficiency varies considerably in type as well as in magnitude. Probably the least serious type is that in which the commissural separation is extended somewhat beyond the normal orificial limits and hence beyond the limits of tendinous support, so that the unsupported partially flexible extremities of the newly created valve "lips" tend to turn backward during ventricular systole.

A somewhat more serious type of traumatic insufficiency is one in which the line of created valvular separation deviates slightly from the true commissural line. This results in a reduction in the support of one leaflet and in a partial turning back of a portion of one valve edge.

An extension of the commissural separation fully into the heart wall may result in division of the atrioventricular annulus fibrosus. This permits a separation of the divided ends of the annulus and hence of the valve edges at this extremity of the orifice which completely prevents approximation during systole.

Still more severe is the regurgitation which results from separation of one leaflet from all of its tendinous supports at one extremity of the valve. This is due to a gross deviation of the line of separation from the natural anatomical commissure, resulting in a "flail" action of the detached end of the leaflet.

Maximal regurgitation is produced by an actual division of the continuity of one of the valve leaflets. This, of course, is greatest when the septal leaflet is divided completely, death usually taking place within fifteen minutes of the injury. While some have pointed out that comparable injury of the mural leaflet is less serious clinically, nevertheless, it is a surgical disaster which at best is attended by great left ventricular enlargement and reduction in cardiac reserve.

There are certain unusual or bizarre types of trauma which may cause regurgitation, such as inadvertent perforation of a cusp with a valvulotome, or rupture of a papillary muscle. They are particularly difficult to correct surgically (Fig. 3, *A-H*).

"*Functional Insufficiency.*"—In a loose way, it is generally recognized that a lack of tone of the myocardium may result in a dilatation or overstretch of the mitral annulus fibrosus. Hence, even a valve with normal leaflet structure becomes unable to hold back the regurgitant stream because of the large size of the atrioventricular canal. Most clinicians think of such regurgitation as essentially transient and reversible, and hence of little serious significance.

No doubt, such reversible annular dilatation may occur during toxic states, in the course of acute infections, and in certain types of congestive heart failure due to loss of myocardial tonicity and hence of the myocardial sphincteric action which normally is exerted about the annulus fibrosus with each ventricular systole.[7] Such insufficiency apparently is transient, disappearing with recovery from the initiating toxemia or congestive failure.

However, there are other forms of annular dilatation which may cause a less reversible "functional insufficiency." These are conditions associated with great left ventricular enlargement. Essential hypertension and aortic valvular disease, either stenotic or regurgitant, not infrequently may be associated with mitral insufficiency of this type. Presumably an improvement in the predis-

posing condition might lead to a reduction in the size of the left ventricle, and hence in the size of the annulus, and thus might cause restoration of full mitral competence.

Apparently the rheumatic process may cause a weakening of the fibrous elements of the mitral valve ring (annulus fibrosus) so that a more persistent

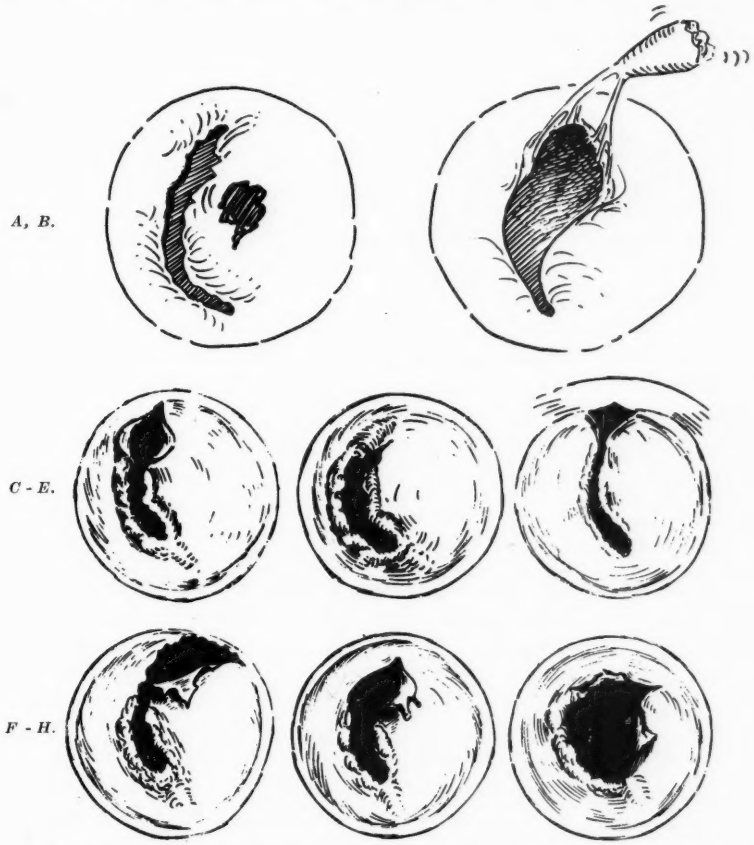
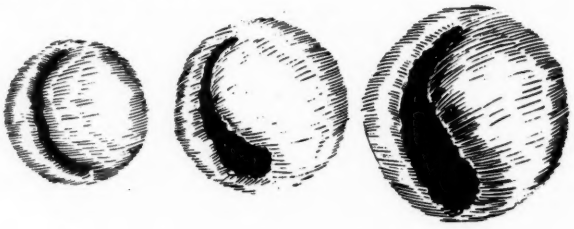


Fig 3.—Traumatic insufficiency. *A*, Gunshot perforation of a leaflet. *B*, Avulsion of a papillary muscle. *C*, Extension of commissural separation beyond normal anatomical limits of support causes limited insufficiency. *D*, Deviation of valvular separation slightly from normal commissural line. *E*, Extreme extension of commissural separation into heart wall may cause division of the atrioventricular annulus fibrosus. *F*, Separation of one leaflet from its heart wall attachment. *G*, "Flail" valve due to separation of a leaflet from its supporting chordae at one extremity of the orifice. *H*, Division of the continuity of a leaflet causes great insufficiency.

or permanent type of annular dilatation results. At autopsy or operation, the left ventricle and atrium are seen to be huge, with little or no constriction between them. During life, a gross systolic thrill may be palpated over the surface of the dilated and hypertensive left atrium, and there may be a visible systolic dilatation or "pulsation" of the latter. Digital intra-atrial examination during life will reveal functioning flexible valve cusps which appear to be en-

tirely normal in structure, a valve orifice of normal size or larger than normal, and a large regurgitant jet of blood which emerges with each ventricular systole from the posterior portion of the valve orifice only. The anterior portion of the valve orifice remains completely competent. In mild cases the regurgitation will be limited to that portion of the orifice which is immediately adjacent to the posterior commissure, but in the more severe cases it may involve up to or even more than half the length of the valve orifice beginning at its posterior extremity (Fig. 4, *A*, *B*, and *C*).



A. *B.* *C.*

Fig. 4.—Functional insufficiency due to annular overstretch. The leak is always posteriorly located. *A*, Normal valve orifice. *B*, Moderate functional insufficiency. *C*, Extreme functional insufficiency.

Progressive Annular Dilatation Due to a Vicious Mechanism.—While undoubtedly there are other pertinent factors, including the condition of the myocardium, especially with respect to rheumatic activity, it appears that once annular dilatation has progressed to or beyond a certain critical point, a vicious mechanism becomes established. Since the only natural mechanisms compensatory for mitral regurgitation are ventricular dilatation and hypertrophy, at least the former phenomenon will take place as soon as insufficiency appears. However, the size of the atrioventricular canal is primarily a function of the size of the left ventricle (unless fibrous contraction of the annulus has been caused by the rheumatic process). Hence, the ventricular enlargement tends to cause further dilatation of the mitral annulus. This produces more valvular insufficiency which, in turn, requires further compensatory ventricular dilatation. This leads again to further annular overstretch, and so on.

Hence, these individuals exhibit progressive and marked cardiac enlargement and continuous decline in myocardial reserve. Some have contended that this phenomenon, especially when it occurs in young persons following a bout of rheumatic fever, is primarily an expression of cardiac deterioration due to an active rheumatic myocarditis. However, the authors feel that the overwhelming mechanical burdens associated with this vicious mechanism if imposed upon even a normal myocardium must lead to its progressive exhaustion and failure. Must not such a process then be even much more deleterious to the diseased heart? Following such reasoning to its logical conclusion and especially in consideration of the overwhelming mortality associated with the conservative treatment of these cases, one might consider the known coexistence of rheumatic activity to be an urgent indication for corrective surgery if such could be performed with a reasonably low risk, rather than a contraindication, as is generally believed by most clinicians. Naturally, common sense must be exercised

in excluding the desperately ill patients whose myocardium would not tolerate even a temporary additional strain and in applying the appropriate preoperative preparatory measures to reduce the operative risk and to enhance the chances for subsequent clinical benefit.

While naturally it might be expected that symmetrical dilatation of the mitral annulus would lead to incompetence greatest in the central portion of the valve orifice similar to the central gap caused by symmetrically stretching a sheet of rubber with a curved incision in its substance, this is seen only when one of the leaflets is found at a higher level than the other in the atrioventricular canal. Here, the valve orifice is arcuate with a central leak. Presumably this lesion is caused by unequal suspension of the two valve leaflets, probably due to unequal shortening of their tendinous supports. In these patients pressure with an intracardiac finger tip against the higher leaflet usually will restore full competence and abolish the regurgitant jet.

In most cases the anterior portion of the valve orifice remains fully competent while the portion near the posterior commissure becomes gaping and leaks. Probably the explanation for this phenomenon lies in the relatively greater amount or extra reserve of valvular tissue which exists in the anterior portion of the valve cone than is found posteriorly. Anatomical studies by the authors upon normal freshly obtained human hearts reveal a constant differential between the amount of apicobasal systolic leaflet overlap (zone of contact) in the anterior portion of the valve and that observed posteriorly. Frequently there is little or no leaflet overlap during systole in the region close to the posterior commissure. Progressive symmetrical dilatation of the annulus will result in increasing separation of the bases of the leaflets, gradually "using up" the available "reserve" valvular tissue and reducing the systolic overlap of their free margins. Obviously, the relatively slight amount of excess valvular tissue which exists posteriorly soon will be exhausted as this process advances, leading to failure of systolic marginal approximation and incompetence, while at least edge contact of the leaflets is maintained anteriorly. A similar mechanism would seem to be responsible in many of the cases of posterior incompetence associated with symmetrical rheumatic retraction of the valve leaflets (Fig. 5, *A-F*).

Fifty-eight of our patients were considered to have this type of insufficiency in pure form, that is, unassociated with any detectable degree of stenosis or organic distortion of the valve leaflets. It must be realized, however, that this type of incompetence may become engrafted upon any of the other types of regurgitation if enough flexibility of the valve structures persists to permit annular dilatation to influence the size of the valve orifice. This is particularly pertinent in cases of operatively produced traumatic insufficiency of significant magnitude where natural attempts at compensation may result in great enlargement of the left ventricle and consequently in the production of significant annular dilatation. In turn, this increases the size of the leak thus setting into play the factors responsible for the vicious mechanism described. However, it may be interrupted by the limits of valve flexibility in these cases. Likewise, "functional insufficiency" due to annular dilatation may be a factor in the over-all picture of mitral regurgitation in many patients with incompletely

rigid valves who initially developed their leak because of rheumatic distortion of one or both leaflets. It is difficult to estimate what part such superimposed "functional insufficiency" may play in the physiologic handicap associated with organic valvular incompetence in any given case. Therefore, we have classified such mixed cases of mitral insufficiency under the organic lesions.

Mitral Insufficiency Due to Acquired Organic Rheumatic Distortion of the Valve Leaflets.—In this condition the mitral leaflets are thickened and retracted due to rheumatic deformity and the pathology tends to resemble that of mitral stenosis with which it practically always is associated. The magnitude of the regurgitation varies greatly, and may be considered, in many cases, to be inversely proportional (reciprocal) to the amount of coexisting mitral stenosis.

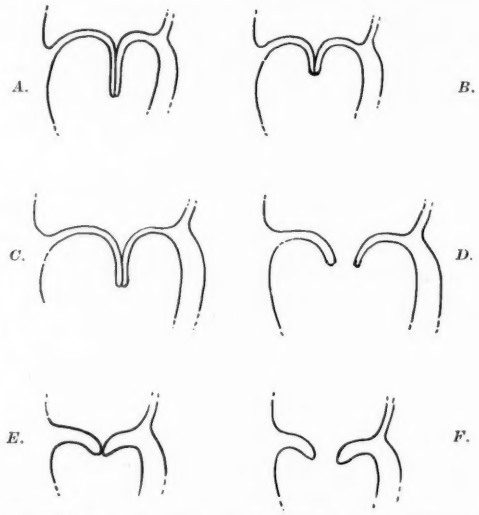


Fig. 5.—Anatomical explanation for posterior localization of incompetence due to annular dilatation and to rheumatic shortening and retraction of leaflet substance. *A*, Long section of normal valve near anterior commissure during ventricular systole. *B*, Same near posterior commissure. *C*, Edge approximation is maintained anteriorly in annular dilatation due to existence of reserve valvular tissue. *D*, Failure of edge approximation in posterior portion of valve in annular dilatation. *E*, Edge coaptation persists anteriorly even with shortened and thickened leaflets. *F*, Near the posterior commissure even slight leaflet retraction causes incompetence.

Indeed, one might presume that the presence of mitral stenosis is to some degree compensatory to mitral incompetence. While it is probably true that initial insufficiency (perhaps due to annular dilatation) may become reduced to a considerable extent by the subsequent development of a stenosing lesion, yet both mitral stenosis and insufficiency repeatedly have been observed by us to coexist in relatively serious form.

Here, as in cases of functional mitral insufficiency, the incompetence is nearly always localized to the posterior portion of the valve orifice. Frequently, the anterior portion of the valve orifice is obliterated by stenotic fusion of the leaflets, involving the anterior peripheral and perhaps most of the central segment of the valve orifice. This results in a "teardrop" shaped orifice with its

blunt rounded extremity adjacent to the posterior commissural tissue. Frequently the substance of the mural leaflet appears to be locally retracted, forming an "incisura." (Fig. 6, *A*, *B*, and *C*.)

There may be two mechanisms by which such posterior incompetence and stenosis may develop. In one, the valve initially may have been subject to annular dilatation and "functional insufficiency" perhaps during the toxic phase of the rheumatic activity. The subsequent development of stenosis follows its usual pattern of centripetally progressing commissural obliteration by cross-fusion of the portions of the leaflets which come into apposition during ventricular systole. However, the failure of leaflet apposition to take place pos-

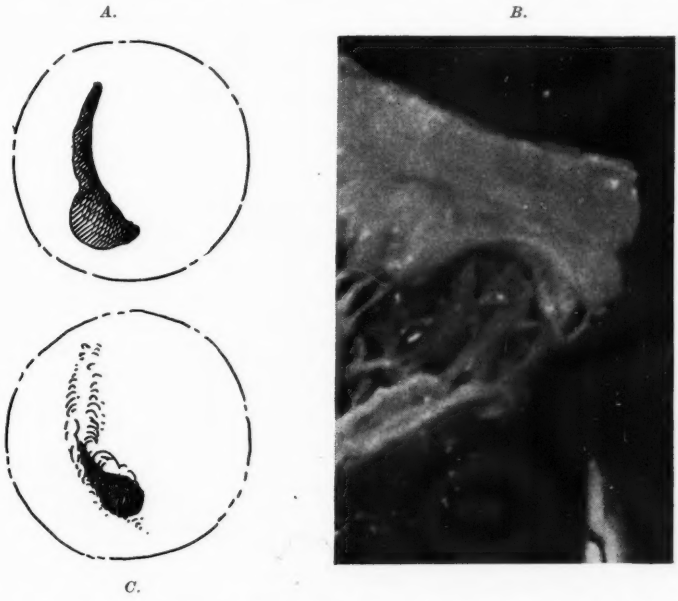
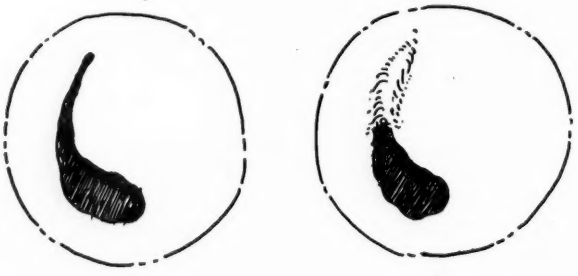


Fig. 6.—Typical incompetence due to rheumatic retraction of the leaflets and associated with a varying amount of stenosis. *A*, Slight associated stenosis. Retraction of posterior portion of mural leaflet causes incompetence. *B*, Photograph of locally retracted mural leaflet showing posterior "incisura." (Reproduced with permission of Dr. Silvio Carvalhal, Dept. of Pathology, São Paulo Hospital, São Paulo, Brazil.) *C*, Predominant associated stenosis ("teardrop" valve).

teriorly even during the height of ventricular systole, effectually prevents even the beginning of valvular adherence in this region. Hence, incompetence and incomplete valvular coaptation persist posteriorly while progressive cross-fusion of the leaflets advances from the opposite valve extremity until a small posteriorly located "teardrop" shaped orifice is created. This is both stenotic and incompetent although the former element usually predominates. Obviously, one may encounter any intermediate stage in the course of this development (Fig. 7, *A* and *B*).

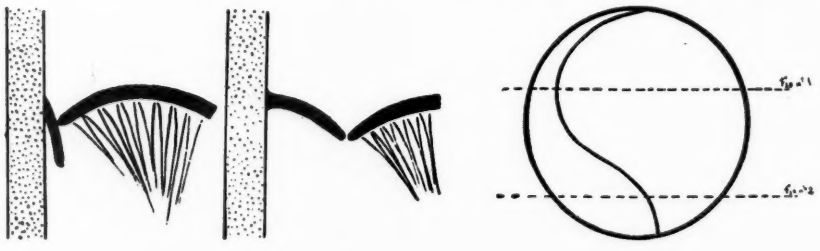
Another explanation for the occurrence of such posterior incompetence relates again to the relative abundance of valvular tissue anteriorly in contrast

to its paucity posteriorly. Hence, symmetrical shrinkage or retraction of the valve edges will produce an exhaustion of the available reserve valvular tissue and failure of coaptation of the edges posteriorly before systolic apposition of the valve edges is prevented anteriorly. Since a stenotic process usually develops simultaneously, again one observes the phenomenon of the stenotic and incompetent posteriorly located "teardrop" valve (Fig. 5, *E* and *F*).



A. *B.*

Fig. 7.—Possible mechanism of development of teardrop valve. *A*, Functionally insufficient valve. *B*, Superimposition of stenosis upon the anterior (competent) portion of the valve. Widely separated posterior portions of leaflets can never become directly fused together.



A'. *A''.* *B.*

Fig. 8.—*A'*, Section through anterior portion of valve. *A''*, Section through posterior. Paravisini interprets the normal mitral valve anatomy in this fashion. The mural leaflet contributes little to competence near the anterior commissure, but greatly posteriorly. *B*, Normal line of mitral valve closure. Hence, specific distortion and contraction of the mural leaflet must cause incompetence posteriorly but not anteriorly. (Courtesy of Medicina Clinica.)

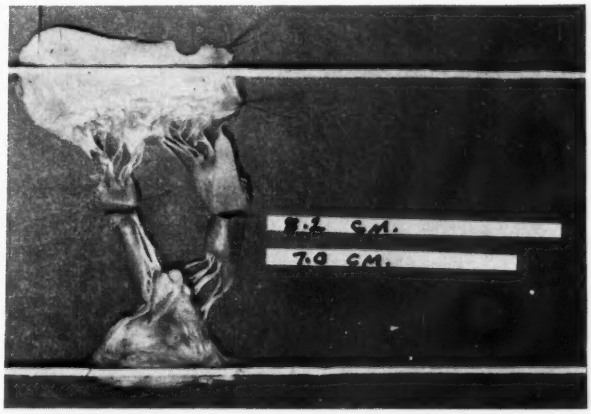
Paravisini and associates[8] have postulated that: (1) the mural mitral leaflet contributes relatively little to valve closure in the anterior portion of the orifice while its importance is equal to that of the septal leaflet in accomplishing closure near the posterior commissure; (2) the rheumatic process has a selectively destructive effect upon the mural leaflet. Hence, reasoning from these two premises it logically may be expected that the localization of mitral valve incompetence will usually be in the posterior portion of the orifice (Fig. 8, *A* and *B*).

Actual measurements of the normal mitral valve leaflets in the fresh or preserved autopsy specimen indicate that the free margin of the septal leaflet characteristically is shorter than that of the mural (Fig. 9, *A*). In five consecutive cases, three of which were preserved specimens and two fresh specimens, the normal mitral valves were found to have the measurements indicated in

Table III. Obviously, since the free margins and the immediately subjacent zone of the leaflets must come into exact opposition during ventricular systole, this must be brought about largely by a reduction in the length of the longer mural leaflet by a "sphincteric" narrowing of the directly attached ventricular wall at the moment of systole. This reduction in the length of the mural leaflet produces a transverse infolding or "wrinkling" of its substance, an appearance which some have interpreted as due to the existence of accessory or intermediate valve leaflets. (Fig. 9, *B*.)

TABLE III. ACTUAL MEASUREMENTS OF NORMAL MITRAL VALVES IN FIVE CONSECUTIVE CASES: THREE PRESERVED SPECIMENS AND TWO FRESH SPECIMENS

	ANNULAR MURAL LEAFLET (MM.)	FREE MURAL LEAFLET (MM.)	ANNULAR SEPTAL LEAFLET (MM.)	FREE SEPTAL LEAFLET (MM.)
1.	40	56	40	52
2.	50	72	50	62
3.	60	68	40	60
4.	40	67	40	55
5.	80	105	40	62



A.

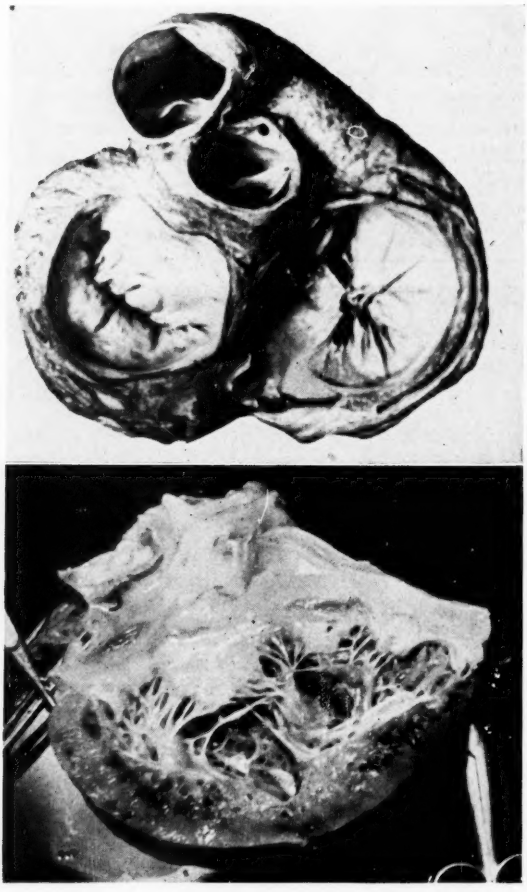
Fig. 9.—*A*, Representative specimen of normal human mitral valve. Note that the free edge of the mural leaflet (upper) normally is longer than that of the septal. *B*, Normal human mitral valve in systole, atrial view. Note infolding or wrinkling in two areas of surface of mural leaflet. Some might erroneously assume this infolding to represent the presence of additional or commissural leaflets; actually, they are due to the necessary infolding of its surface caused by the discrepancy in the lengths of the free margins of the two leaflets when the contraction of the left ventricle reduces the length of the mural leaflet to fit the free margin of the septal. (Courtesy Prof. Harald Okkels, Institute of Human Aanatomy, Copenhagen, Denmark.) *C*, Photograph of a human mitral valve showing true accessory or commissural leaflets. This variant is believed to occur in 5 per cent of human cases. (Courtesy Dr. Silvio Carvalhal, Dept. of Pathology, São Paulo Hospital, São Paulo, Brazil. *D*, Photograph of bull heart showing quadricuspid structure which is normal in this species. D_1, Atrial aspect. D_2, Ventricular aspect.

Actually such accessory leaflets are rather rare in human beings. Rusted, Scheifley, and Edwards[9] have estimated that they may occur in perhaps 5 per cent of patients. Fig. 9, *C* illustrates not only the existence of an accessory leaflet in a human mitral valve but also a rheumatic "incisura" of the mural leaflet, which has the effect of lengthening the free margin and thus causing insufficiency in part due to leaflet marginal disproportion.

The bull heart characteristically presents two large accessory commissural leaflets in addition to the two major ones (Fig. 9, D_1 and D_2).

Naturally, there are many variations in the kind and magnitude of mitral regurgitation observed when it is due to thickening and distortion of the leaflets. In some, the valve will have become converted essentially into a fibrotic or

B.

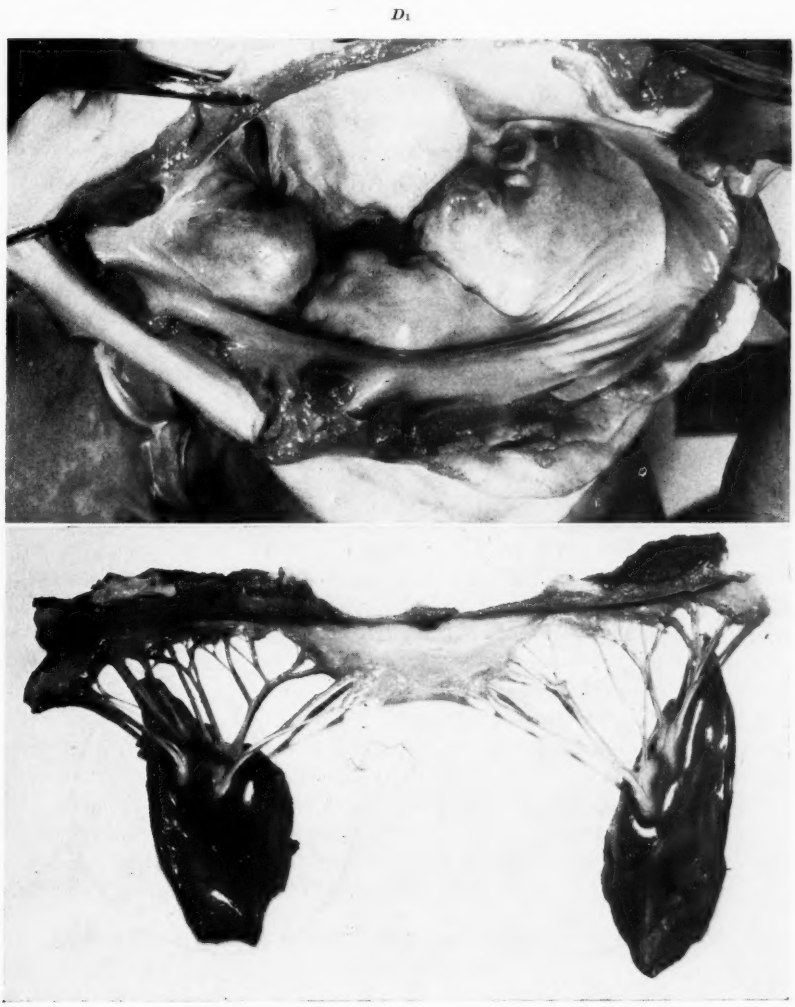


C.

Fig. 9, *B* and *C*. *(For legend see page 563.)*

calcific plaque with a round or ovoid central opening. While such an opening ordinarily is small and stenotic, occasionally one is seen which is large enough to admit a man's thumb. Since such severe regurgitation cannot long be tolerated even by the strongest heart with the healthiest myocardium, these indi-

viduals have a relatively short life expectancy. In an occasional patient the valve structures may become adherent directly to the adjacent ventricular wall, producing insufficiency. (Fig. 10, *A*, *B*, and *C*.)



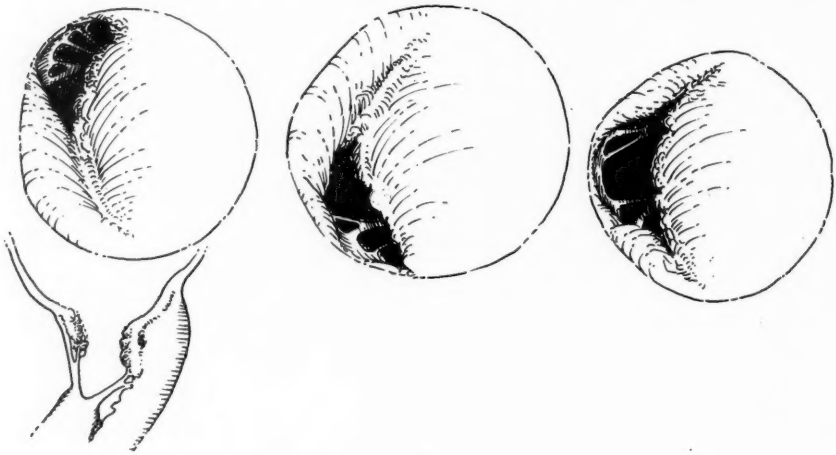
*D*₁

*D*₂

Fig. 9, *D*₁ and *D*₂. (*For legend see page 563.*)

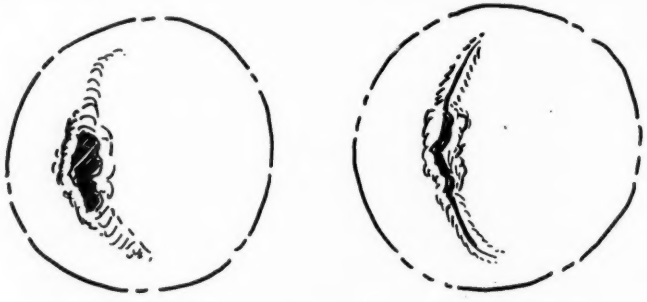
There are many patients who present features of both functional and organic insufficiency in varying proportions. These individuals may have relatively little associated over-all reduction in the size of the valve orifice (little associated stenosis) but the thickening and distortion of the leaflets are typical of rheumatic disease.

Another form of insufficiency which must be included in this classification is that which is associated with irregularity of the thickened valve edges which renders accurate apposition impossible. Such incompetence usually is found in association with a stenotic hard valve, often with extensive calcification of the leaflets. This type of incompetence nearly always is of relatively small magni-



A. *B.* *C.*

Fig. 10.—Mitral insufficiency due to direct adherence of papillary system and/or actual valve substance to the ventricular wall. *A*, Adherence of valve structures to ventricular wall in the region of the anterior commissure, and *B*, in the region of the posterior commissure. *C*, Adherence of the central portion of the mural leaflet or its supporting structures to the lateral ventricular wall.



A. *B.*

Fig. 11.—Insufficiency associated with stenosis in which the irregular hard margins of the often calcified valve may become more accurately apposed after performance of a proper commissurotomy. *A*, Predominantly stenotic but also incompetent valve. *B*, Improvement in competence after commissurotomy.

tude and hence usually may be ignored clinically. It frequently becomes improved by readjustment of the valve coaptation after the performance of an effective commissurotomy (Fig. 11, *A* and *B*).

It will be observed that all forms of mitral insufficiency except that due to unequal papillary suspension and those with direct fusion of the leaflet to the

ventricular wall present one feature in common—a relative or actual lack of valve substance. Hence, it would seem that any corrective measures effective for them must be designed to supply this lack of available substance.

The treatment of valvular incompetence due to differential shortening of the chordae suspending the respective leaflets would seem logically to be such as would lengthen the shorter chordae or shorten the longer ones. The former concept has been expressed both by Dodrill[10] and by Kay.[11] They have attempted to detach acquired adherence between the papillary system and the ventricular wall by transvalvular digital separation.

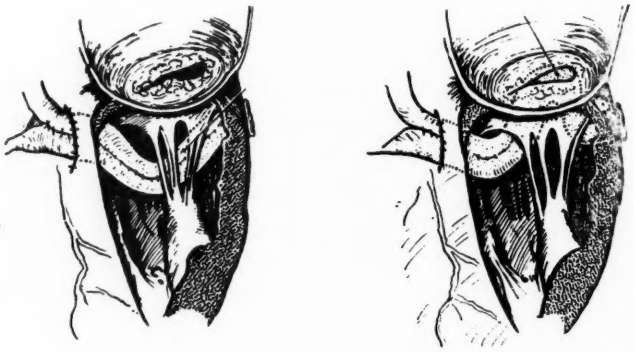
We[2] have attempted to advance the attachment of the leaflet which is displaced farther toward the atrium during ventricular systole by using one or more transventricularly placed pericardial sutures to draw the free edge of the leaflet somewhat farther toward the ventricular apex. This has worked reasonably well in this type of case. Recently one of us (W. L. J.) has modified this technique so that the leaflet is not suspended by a suture about the free edge (which must create some irregularity upon the line of leaflet coaptation and hence must create some new incompetence of a different type). The improved technique entails perforation of the higher leaflet by transventricular passage of a threaded probe. The left index finger placed within the left atrial chamber guides the tip of the probe which subsequently follows the course of the finger to emerge from the opening in the tip of the left auricular appendage. The suture it bears is grasped and the probe is removed from the heart. A knot is tied in one end of a strip of pericardium 2.5 cm. wide and the other end is cut in tapering fashion. The pointed end is attached to the extremity of the transcardiac suture which emerges from the auricular appendage. Traction upon the extremity emerging from the ventricular wall causes the pointed end of the pericardial strip to enter the heart, to traverse the perforation in the valve, and to emerge from the puncture in the ventricular wall. The "knot" enters the left atrium and, being too large to penetrate the perforation in the valve cusp, comes to lie upon its atrial aspect, somewhat lateral to the free margin. Variable traction may be made upon the emerging ventricular extremity of this "artificial chorda tendinea" until maximal improvement in the regurgitation is determined by the intracardiac finger. The extremity of the pericardial ribbon is then secured at that tension or at an estimated safer tension to the ventricular epicardium by direct suturing. (See Fig. 34.)

MEASURES DESIGNED TO OVERCOME AN ACTUAL OR RELATIVE LACK OF VALVULAR SUBSTANCE

Murray[12] originally suggested the transventricular placement of a loosely swinging tissue hammock composed of an inverted vein segment, strengthened by a centrally placed strip of fascia or a tendon. Bailey and associates[1] modified this technique, applying a pedicled tube prepared from pericardium loosely across the ventricular aspect of the incompetent mitral orifice. It was reasoned that such tissues loosely placed on the ventricular aspect of the mitral valve would tend to swing into the orifice, tamponading it, during each ventricular systole. During diastole it would fall away from the mitral aperture to permit ventricular filling (Fig. 12, *A* and *B*).

Such "hammock" valves initially worked well mechanically. However, their placement resulted in a high operative mortality (38 per cent in 52 cases). This was, in part, related to the trauma to the already overburdened and dilated left ventricle which tends easily to go into a state of standstill or ventricular fibrillation. Part was due to an initial tendency toward swelling of the graft which, on a few occasions, due to its increased bulk, produced significant displacement of the ventricular contents. More serious was the not infrequent production of a poorly understood hemorrhagic syndrome in which all cut surfaces would ooze fatally, in spite of replacement therapy or reoperation.

These grafts become replaced partially by fibrous connective tissue and shrink to perhaps two-thirds of each former dimension. Hence, the newly created valvular action becomes abolished in time. For these various reasons the procedure of "transventricular pericardial tamponade" was finally abandoned as clinically unsatisfactory as well as operatively dangerous.



A. *B.*

Fig. 12.—Transventricular placement of pedicled pericardial hammock in the treatment of mitral insufficiency. *A*, Position of graft during diastole. *B*, Position during systole. Leak is completely tamponaded.

Reconsideration of the whole problem and its various related facets led inevitably to contemplation of the clinical experience of others as well as our own group that opening of only one obliterated commissure usually is sufficient to produce great clinical benefit in patients with mitral stenosis. In our entire series of patients with mitral stenosis, treated by commissurotomy alone, we have found that surgical valvular separation was limited to the anterior commissure alone in 64.9 per cent of the cases and to the posterior commissure alone in 1.9 per cent. This final opening would represent, therefore, only the transverse central channel of the original mitral orifice, plus one of the peripheral (designated lateral by Brock[13]) sloping segments of the mitral orifice. The residual flexibility of the valve structures in these individuals is usually sufficient to permit a satisfactory eccentric type of valve function as well as an adequate passageway for blood flow. If such a valve aperture in which the posterior peripheral segment remains fused is adequate to restore the patient's clinical health following commissurotomy for mitral stenosis, should not an identical

aperture be equally adequate to bring about the health of a patient who has undergone an operation for the surgical correction of mitral insufficiency?

An affirmative answer obviously being appropriate to this question, an effort was made to devise a means of restoring the valve aperture in cases of significant mitral regurgitation to one in which the full-sized transverse portion of the central channel, and one full-sized peripheral segment can be assured,

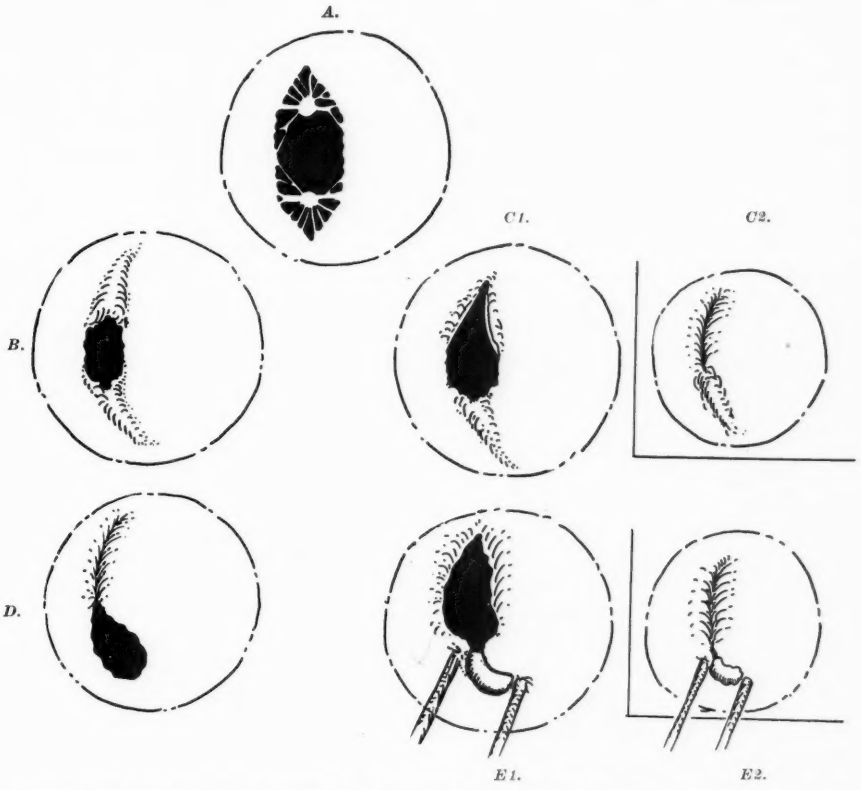


Fig. 13.—*A*, Anatomical division (Brock) of normal mitral valve orifice into three segments, a transverse central segment, and two lateral or peripheral segments. Top view of valve. *B*, Mitral stenosis with obliteration of both peripheral segments. *C*, Anterior commissurotomy alone, restoring orificial size to that of the central and one peripheral segment, establishes clinical health: *1*, ventricular diastole; *2*, ventricular systole. *D*, Mitral insufficiency limited to posterior peripheral segment. *E*, Suture obliteration of posterior peripheral segment with maintenance of central and one peripheral segment should provide adequate orificial size and function equal to that after commissurotomy. If anterior commissurotomy is necessary for associated stenosis, it should be performed before valvular suturing: *1*, ventricular diastole; *2*, ventricular systole.

while the opposite incompetent peripheral segment is obliterated by surgical suturing. Since the posterior peripheral segment is practically always the incompetent one, this is the one chosen for surgical obliteration. While the anterior peripheral segment often is adequately open as in insufficiency due to annular dilatation, all cases with coexistent mitral stenosis will require prior valve enlargement by anterior commissurotomy (Fig. 13, *A-E*).

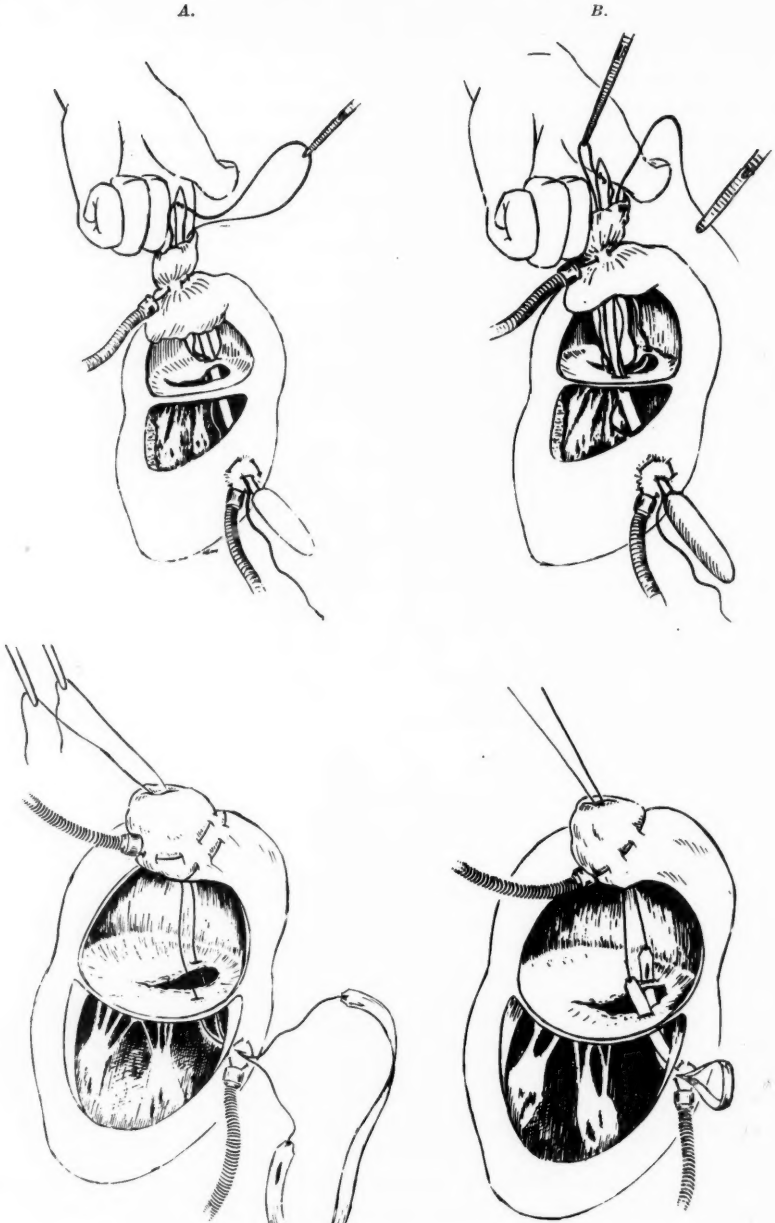


Fig. 14, *A* to *D*. (*For legend see opposite page.*)

The accomplishment of this objective, the surgical obliteration of the incompetent posterior peripheral segment of the valve orifice, while assuring adequately open and functional transverse and anterior peripheral segments, would be greatly facilitated by an open technique under direct vision. Such a technique is not yet practicable, but closed methods are already available which render this procedure fully feasible at this time.

At first, a simple method of suturing the valve was worked out in which a threaded probe was passed through the apex of the left ventricle and, guided by the intracardiac finger, was caused to penetrate the appropriate valve leaflet at the selected point. The probe tip then followed the intracardiac finger until it emerged from the opening in the left auricular appendage. The suture material on one side of the probe was grasped and held while the probe was drawn back into the heart and disengaged from the valve. Then it was redirected to penetrate the other valve leaflet at a selected site. Again, the probe tip followed the finger to emerge from the left auricular appendage. Now, the opposite side of the suture was grasped and held. After withdrawing the probe from the heart, the two leaflets were found to be separately penetrated by the two strands of silk suture, an end of each emerging from the ventricular incision, the opposite emerging from the auricular.

As previously described,[2] initially it was considered satisfactory to attach the extremities of a living tissue strip to the ends of the suture material emerging either from the ventricular or the atrial incision and to draw them by traction upon the opposite ends of the suture material, into the heart, through the perforations in the valve, and finally out of the opposite chamber of the heart. By creating an "eye" in one extremity of the pericardial strip and passing the opposite one through it, a "noose" was formed which could be tightened to any desired degree by simple traction upon the intact extremity. The intracardiac finger was able to appreciate the effect this tightening of the noose had upon the regurgitant jet and hence upon the incompetence itself. In some cases more than one such suture was used (Fig. 14, *A-D*). This procedure has been designated by the term commissurorrhaphy.

It would seem at first glance that such a procedure would fulfill all the requirements for the surgical correction of mitral insufficiency in the average clinical case. The posterior incompetent peripheral segment of the orifice may be obliterated by approximation of the valve edges in this region. This abolishes the regurgitation or, at least, diminishes it greatly. On the other hand, maintenance of the full aperture of the anterior peripheral and the central transverse segment of the valve orifice would seem to ensure a suitable passageway for atrioventricular blood flow. In individuals with significant coexistent mitral

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Fig. 14.—Valvular suturing by transventricular passage of a threaded probe. This technique proved dangerous in application. *A*, Transventricular passage of threaded prober, piercing a valve leaflet and emerging from auricular appendage alongside operator's intracardiac finger. One side of suture is grasped and held. *B*, Probe is partially withdrawn to become disengaged from ventricular aspect of valve. By immediate redirection it pierces the opposite leaflet and again emerges from the appendage. The opposite side of suture is grasped and the probe is withdrawn. *C*, After the probe is withdrawn, two sutures completely traverse both chambers of the left side of the heart, each penetrating a valve leaflet. Either pair of ends may be attached to the extremities of a prepared pericardial ribbon. *D*, Replacement of the sutures with a strip of living tissue (pericardium) for atrial (or ventricular) suspension. (Bailey et al., courtesy S. Clin. North America, W. B. Saunders Co.)

stenosis specifically involving the anterior portion of the valve, this element of impaired valve function must be overcome by a preliminary commissurotomy procedure before applying the valve approximating sutures.

DISCUSSION

Unfortunately, several untoward features were associated with this approach. In a series of 72 patients operated on by this general technique or some close modification, there were 20 hospital deaths, a mortality of 27.8 per cent. While this might be explained in part by the fact that this was a pioneering undertaking with a constantly changing developmental technique, and in part by the extremely poor condition of many of the patients chosen for this type of surgery, it became increasingly evident that the procedure was inherently too dangerous, and that it presented some other very practical drawbacks.

One of the most obvious causes of mortality was the sudden onset of ventricular fibrillation which tended to occur especially during the manipulations of the left ventricle. It seems to us that with the dynamically balanced circulation which these individuals exhibit, in which an appreciable and perhaps major portion of the left ventricular output traverses the incompetent mitral orifice to enter the relatively low-pressure left atrial chamber while the remainder is expelled into the high-pressure aortic lumen, a relatively slight depression in left ventricular vigor will result in a much greater proportional decline in aortic output since the majority of the reduced ventricular output will then tend preferentially to enter the low-pressure left atrium (Fig. 1, *A* and *B*). Thus, even a slight fall in left ventricular output may precipitate a relative inadequacy of coronary filling and flow, and hence initiate ventricular fibrillation.

It would seem that probe puncturing of the left ventricular wall, the repeated manipulation of the probe within the ventricle, the inevitable momentary distortion of the functioning valve, and the associated blood loss, all tend to produce ventricular fibrillation.

Autopsy studies in several patients who died in the hospital showed that the pericardial suture or sutures had cut through the substance of the valve in several instances, presumably because of too great tension by the tough pericardial tissue in the presence of relatively fragile leaflets. Of course, this occurrence was accompanied by a restoration or worsening of the pre-existing valvular incompetence (Fig. 15). In another patient, the ventricularly suspended pericardial strip became broken (Fig. 16).

Reflection suggested that the excessive pericardial strip tension was in part due to attachment of the free end to the ventricular epicardium since it was not observed when the pericardial strips were brought out of the relatively more yielding auricular appendage and were simply attached to its stump. Moreover, it will be perceived readily that at the height of ventricular systole the mitral leaflets are forced backward, away from the site of ventricular suspension by any pericardial noose-suture combination, thus increasing the tension

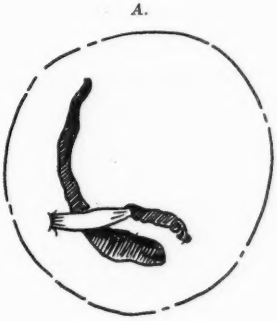
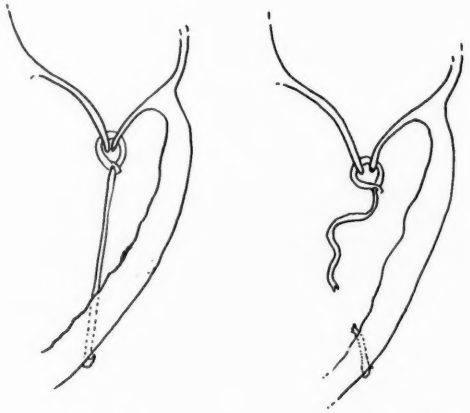


Fig. 15.—An ugly possibility in noose-type suturing with ventricular suspension is tearing of the substance of the valve leaflets (if not heavily fibrosed) by repeated traction of the pericardial sutures during systole. *A*, Diagrammatic representation. *B*, Actual photograph of valve three months after noose suturing and ventricular suspension. *C*, Expression of fluid from ventricle through lacerated valve substance in same case. (Courtesy Dr. Silvio Carvalhal, Dept. of Pathology, São Paulo Hospital, São Paulo, Brazil.)

upon the valve leaflets. If they have not become reinforced by a fairly marked degree of fibrosis, the incessantly repeated pull of the noose must certainly tend to tear through the valve substance.

On the other hand, suspension of the noose from the appendageal stump would seem to diminish this risk. Since the leaflets tend to approach the atrial chamber during ventricular systole, the "noose" tension would become diminished at this time. Also, it would seem that the relatively yielding attachment to the atrial stump would provide a cushioning effect against any very sudden increase in tension during systole. For these reasons, ventricular suspension, at least when a "noose" is used to approximate the valve edges, has been abandoned.



A. *B.*

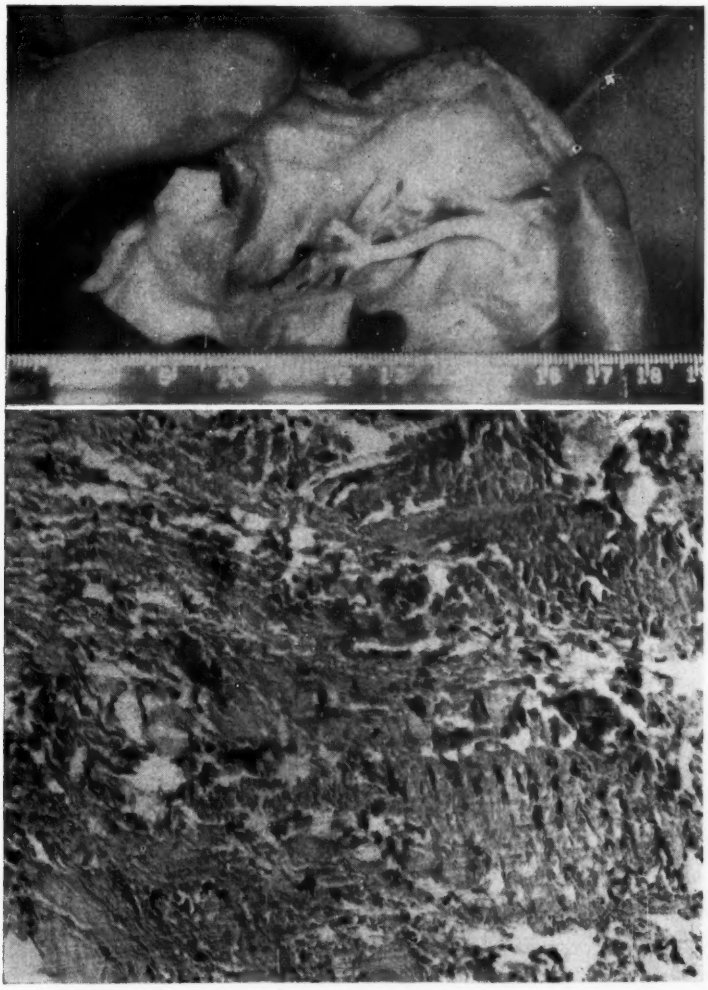
Fig. 16.—Disadvantage associated with suspension of sutured valve from the ventricular wall. Incessantly repeated strain from ventricular contraction may cause rupture of the pericardial strip. This is known to have occurred in one of the author's cases. *A*, Valve sutured. Ventricular suspension by noose technique. *B*, Dissolution of continuity of strip (six months after placement).

Of course, if noose tension were to become reduced at the moment when intraventricular tension is at its maximum, it is probable that regurgitation might not be as fully restrained as it would be were the tension fully maintained at that time. In view of our previous experience with the ultimate fate of transventricular pericardial hammocks, it was presumed that eventual shrinkage and fibrosis of the graft and the late extension of the fibrosing process into the adjacent valve leaflets would result in sealing of the posterior peripheral segment of the valve orifice by a "welding" or fusing of the adjacent valve leaflets together as well as to the tissue suture. That this does occur when the suture graft is large, is illustrated in Fig. 17, *A* and *B*. However, if the suture material is thin, its replacing fibrous tissue may become "stretched out" and functionless as shown in Fig. 18, *A*.

However, a most important factor which we have not yet considered is that in most instances of mitral regurgitation there is an actual or relative lack of sufficient valvular tissue in the posterior portion of the orifice to permit edge

approximation of the leaflets except with the use of considerable tension upon the suture-graft. Hence, unless these leaflets have become strengthened by fibrosis they will not tolerate sufficient noose tension to restore full competence.

A.



B.

Fig. 17.—*A*, Gross appearance of mitral pericardial suture 4½ months after placement. Note "welding" together of graft and valve edges. Anterior two-thirds of valve orifice remains flexible and functional. *B*, Photomicrograph showing partial conversion of pericardium into fibrous connective tissues.

In other words, when there is a dearth of available valvular tissue, it seems necessary to supply it either by a grafting process or by the use of a prosthesis of some kind. The inherent disadvantages associated with the use of prostheses

inserted within the circulatory system, and the specific and overwhelming dangers implied in their placement within or near the mitral orifice have deterred us from pursuing this line of thought.

A.

B.

Fig. 18.—*A*, Very slender pericardial suture placed two years before death. Note stretching out. *B*, Photomicrograph of same transformed graft illustrating fibrous change.

Before considering the placement of tissue grafts within the cardiac chambers, it would be well to establish the ultimate fate of pericardial tissue so placed.

Glover, Henderson, and associates,[14] in 1953, after a series of experiments in which free and pedicled pericardial tissue was placed across the left ventricu-

lar chamber in dogs, came to the conclusion that these grafts became degenerated and replaced by shrunken fibrotic cords (Fig. 19, *A-D*). Moore and Schumacher,[15] in 1953, came to somewhat similar conclusions in a similar study (Fig. 20, *A* and *B*). Bailey, Lacey, and associates,[16] in 1952, had shown the

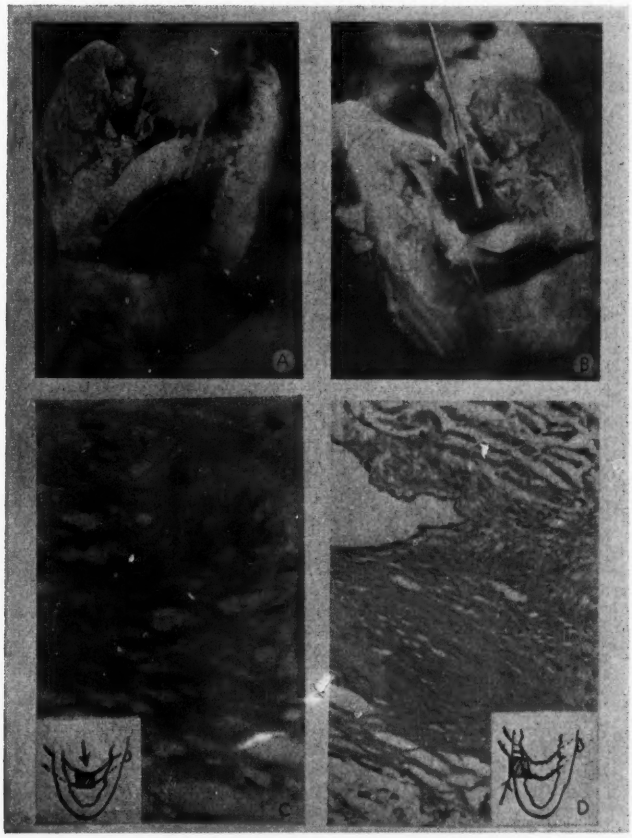
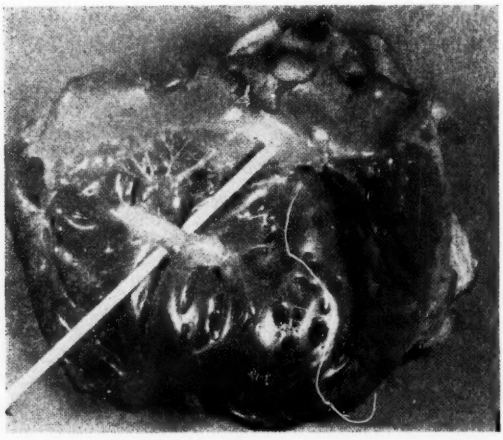


Fig. 19.—*A* and *B*, Experimentally placed pericardial grafts in dogs. *C* and *D*, Photomicrographs of sections of grafts. (Glover, Henderson, et al., Surgical Forum, 1952, Am. Coll. Surg. Reproduced with permission of W. B. Saunders Co.)

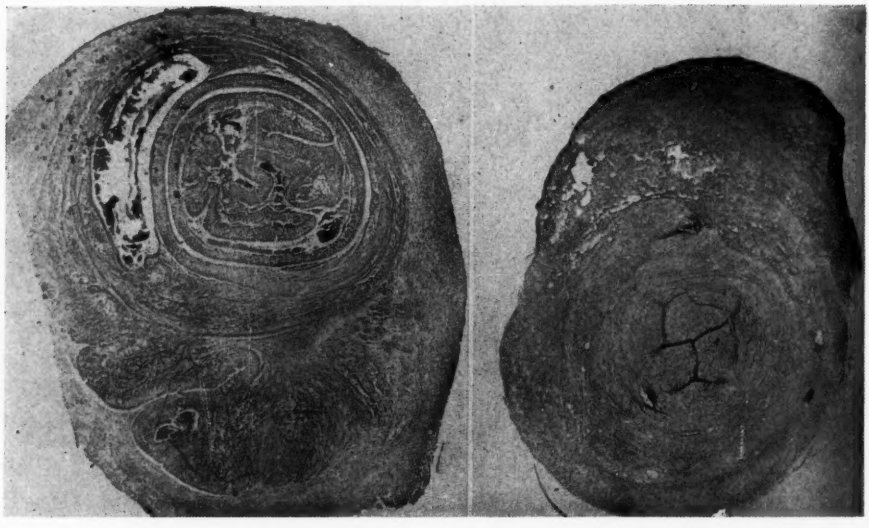
early fate of such pericardial tissue and had pointed out the futility of using sheet pericardial material for the purpose of covering a septal defect (Fig. 21, *A* and *B*).

Today, with the availability of human patients with pericardial grafts and sutures placed as long as four years ago, we are in a position to clarify these doubts. Initially these grafts swell remarkably (Fig. 22). On four different occasions, one of us (C. P. B.) has been able to examine digitally, during life, transventricular pericardial pedicled grafts placed 12 to 36 months

earlier. In three instances, the graft had become shortened and stiffer, in two of them being stretched tightly across the left ventricular chamber but away from the valve. In one, the graft had become sealed to the valve orifice, completely



A.



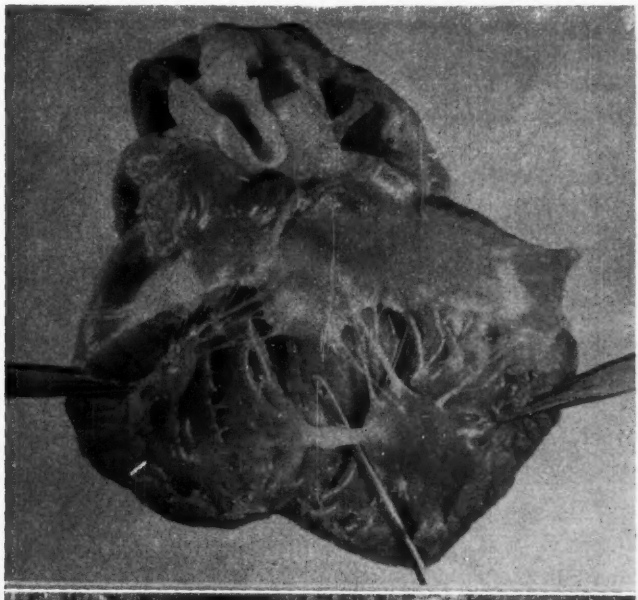
B. *C.*

Fig. 20.—*A*, Photograph of pericardial graft placed in dog's heart fourteen weeks previously. *B*, Photomicrographs of sections through grafts 1½ weeks after placement. *C*, Photomicrographs of sections through grafts fourteen weeks after placement. (From Moore and Schumacher: Surgery 33:173, 1953.)

restraining all regurgitation but producing a high degree of mitral stenosis. It was possible to perform an instrumental "commissurotomy," cutting the graft away from the septal leaflet, thus producing good valve function as well

as relieving the stenosis and without restoring the original incompetence. The graft remained attached to the mural leaflet, adding a considerable amount of new living tissue to its retracted free edge (Fig. 23, *A* and *B*). From this experience, came the concept of deliberately performing selective plastic grafting

A.

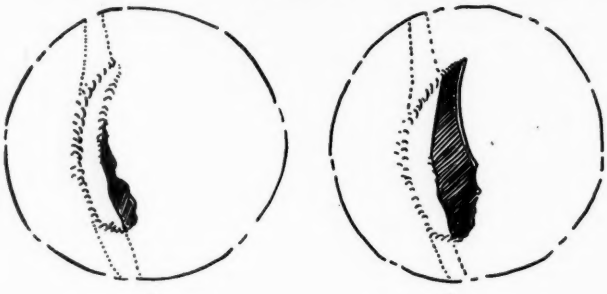


B.

Fig. 21.—*A*, Photograph, and *B*, photomicrograph, of shrunken sheet graft of pericardium three months after placement in dogs. (From Bailey, Lacey, et al.: Ann. Surg. 136:919, 1952.)

to the edge of the mural leaflet. In the fourth case, the graft was somewhat smaller in caliber but appeared to maintain all of its original flexibility and length.

Fig. 22.—Appearance of pericardial hammock applied in a human patient two days after placement.

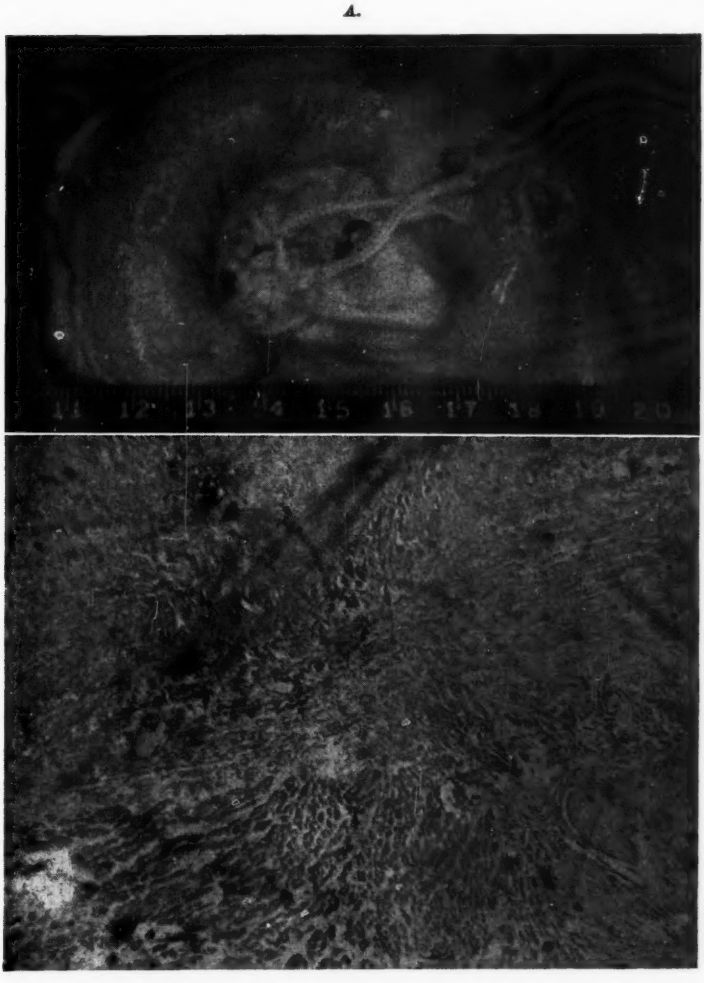


A. *B.*

Fig. 23.—*A*, Ten months after placement of transventricular pericardial hammock for mitral regurgitation, at reoperation for symptoms of mitral stenosis, the somewhat shrunken graft was found adherent to the ventricular aspect of the valve edges. *B*, "Commissurotomy" resulted in mobilization of septal leaflet from graft with relief of stenosis and without restoration of regurgitation. Graft remained attached to free edge of mural leaflet adding needed tissue in its retracted portion.

In addition, four patients have come to autopsy four months to two and one-half years following placement of free intracardiac pericardial sutures. One of these is illustrated in Fig. 24, *A* and *B*. Two others were shown in Figs. 14, *A* and *B* and 18, *A* and *B*, respectively. In all instances, the graft was replaced largely by fibrous connective tissue. The thicker grafts have shown

shrinkage and contraction. The thinner graft was elongated and stretched out. This is in contrast to pedicled grafts which retain much of their histologic structure and characteristically maintain most of their original bulk.



A.

B.

Fig. 24.—*A*, Appearance of pericardial suture thirty months after placement. *B*, Photomicrograph of the same.

In summary, we may say that pericardial sutures or grafts placed within the free cardiac chambers do not die or disintegrate. They tend to become replaced partially by fibrous connective tissue and to shrink both in length and in diameter. The latter becomes two-thirds to one-half of the original

measurement, although it must be remembered that initially (twelve hours to ten days) these grafts may swell to two to four times their normal diameter and hence may encroach seriously upon the capacity of the chamber in which they are placed.

The application of these observations to the consideration of further use of such grafts is obvious. They cannot be depended upon for prolonged flexible movement. They can be used as suture material or as grafts to add fibrous tissue to the structure to which they are applied. While they shrink significantly, the larger grafts remain of appreciable size at least for a period of two years, and presumably indefinitely. Pedicled grafts tend to maintain a fair size and some flexibility permanently.

Fig. 25.—In patients with valve orifices of limited attainable size, direct cross-suturing of the valve leaflets may result in the creation of significant stenosis.

CREATED MITRAL STENOSIS

Another very pertinent point is concerned with the possible surgical production of an incapacitating degree of mitral stenosis. While the likelihood of such an occurrence would seem to be extremely remote in patients with widely open valve orifices incompetent chiefly or entirely because of annular dilatation, significant mitral stenosis was produced in several patients with valve openings which were of borderline size even after the performance of an anterior commissurotomy. (Fig. 25.)

This experience led to a modification of the concept of actually suturing together the posterior portions of the leaflets in any case where there was even the slightest doubt as to the adequacy of the eventual size of the opening. Instead, a heavy suture of pericardium was placed from the valve substance

in the region of the posterior commissure to the free margin of the mural leaflet usually at about its midportion. This amounted to an attempt to obliterate the exaggerated concavity of the mural cusp due to fibrosis and retraction

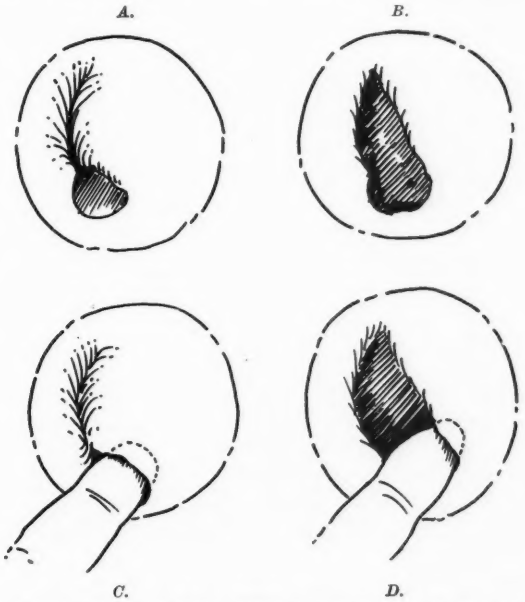


Fig. 26.—In mitral valves with the usual posterior type of regurgitation, sufficiency can be restored temporarily by the placement of the tip of the operator's index finger into the incompetent portion of the orifice. This amounts, in effect, to the temporary replacement in this region of the actually or relatively lost valvular substance. *A*, Posteriorly incompetent mitral valve during ventricular systole, whether caused by annular dilatation or intrinsic (rheumatic) shrinkage of the leaflets. *B*, Same valve during diastole, provided the septal leaflet retains mobility. *C*, Blockage of incompetence by the mass or bulk of the operator's finger tip. Ventricular systole. *D*, Adequacy of aperture and valve function provided by residual mobility of leaflets. Ventricular diastole.

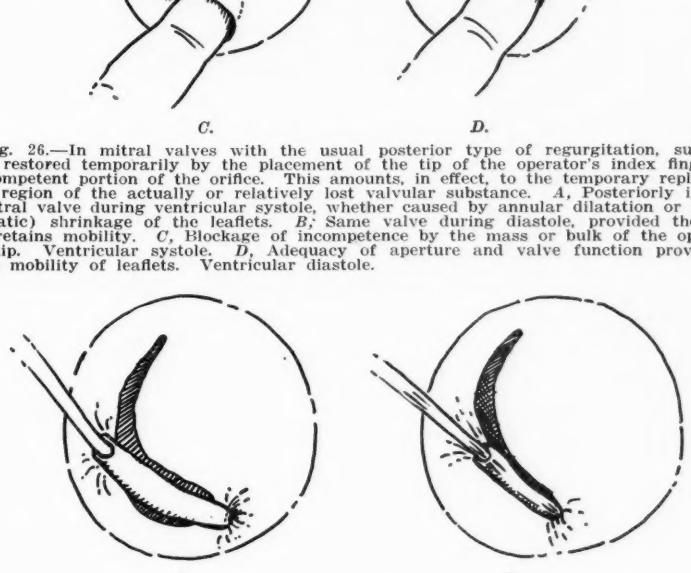


Fig. 27.—Addition of needed tissue by applying a thick pericardial strip to the deficient posterior leaflet. *A*, Simple addition of tissue to incompetent portion of the valve. *B*, Eventual contraction of graft tissue will reduce the area of incompetence.

in this portion of its margin by adding new grafted tissue to its free edge. This application of new tissue permitted unhampered movement of the septal leaflet while the valve opening remained nearly as large as before. In effect,

the result aimed at was similar to that accomplished temporarily by placing the intracardiac finger tip within the posterior incompetent portion of the valve orifice (Fig. 26, *A-D*). Such digital tamponading of the incompetent posterior portion of the valve may restore complete competence, abolishing the regurgitant jet, without causing significant obstruction of the valve channel. Unfortunately, the transventricular suturing technique limited the size of the pericardial suture which could be used conveniently. Hence, the regurgitation merely was reduced in many of these cases and not completely abolished. The clinical result obtained in these individuals was about proportional to the technical achievement with regard to valve function. (Fig. 27, *A* and *B*.) Table IV illustrates the physiologic changes in E. N. 10 months after performances of such a commissurorrhaphy.

TABLE IV. PHYSIOLOGIC DATA ON CASE E. N.

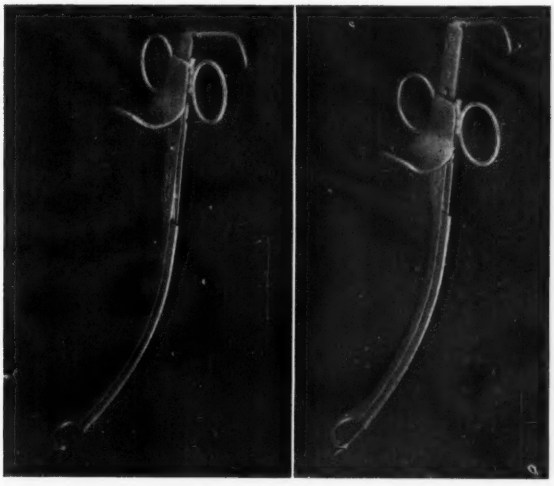
	PREOPERATIVE (12/9/53)		POSTOPERATIVE (9/9/54)	
	REST	EXERCISE	REST	EXERCISE
1. Oxygen consumption (c.c./min.):	216.8	-	202.1	400.9
2. A-V oxygen difference (vol. %):	4.1	-	4.0	5.8
3. Cardiac output (1/min.):	5.3	-	5.1	6.9
4. Cardiac index (units/min./M²BS):	3.9	-	3.4	4.6
5. Pressures (mm. Hg):	—	-	-	-
a. "Pulmonary venous capillary"	(30)	-	(13)	-
b. Main pulmonary artery	60-75 / 20-30 (43)	-	32-38 / 0-8 (27)	50-54 / 10-12 (27-30)
c. Right ventricle	—	-	34 / 0	-
d. Right atrium	—	-	(-2)	
e. Brachial artery	120 / 70 (80)	-	100 / 88 (64)	
f. Peripheral oxygen saturation (%):	90.9%	-	92.9	94.7
g. Pulmonary arteriolar resistance (dynes sec. cm.$^{-5}$):	—	-	220	-

Figures in parentheses refer to mean pressures.

SUTURING OF THE VALVE BY THE AURICULAR APPROACH

In the summer of 1953, one of us (W. L. J.) developed an instrument embodying the principle of the "looper" which is used in the hosiery industry to sew the toes on ladies' stockings. This instrument was designed to permit suturing of the mitral valve by a purely transatrial approach, being inserted along the operator's intracardiac index finger through the opening in the left auricular appendage. Subsequent modifications of this instrument notably by

two of us (W. L. J. and A. E. B.) have culminated in the "mitral stitcher" (Fig. 28, *A* and *B*) which is a reasonably satisfactory instrument for this purpose. Since this method of valvular repair has become possible, very striking improvement in the operative course and mortality and in the clinical results have become apparent in these cases.



A. *B.*

Fig. 28.—Improved mitral "stitcher": *A*, open; *B*, closed.

APPLICATION OF A TRANSVALVULAR PERICARDIAL DIAPHRAGM

Most of those who have investigated the problem of mitral insufficiency clinically[1, 2, 17, 18] have come essentially to the same conclusion, that living or prosthetic tissue must be supplied in such a way as to add new substance to the free edge of the actually or relatively retracted posterior portion of the mural leaflet. It was pointed out by one of us (H. T. N.) that a nonmobile pericardial tube placed diagonally across the posterior half of the valve orifice in close proximity to this cusp would accomplish the desired purpose. Furthermore, the very secure attachment of such a graft at the sites of ventricular wall penetration would obviate the risk of tearing the valvular substance when it is thin and nonfibrotic. Even complete retraction of the leaflet edge or commissural tissue against the inner ventricular wall (which would render accurate valve cusp penetration extremely difficult) would not hamper the placement of such a transvalvular pedicled tube or "diaphragm." Since the tube may be caused to traverse the ventricular wall in an oblique fashion, it can be placed at any level up to the actual valve ring without endangering the circumflex coronary artery which lies upon the latter. Should one such graft prove incapable of blocking all of the regurgitation, a second one may be applied alongside it by

parallel penetration of the ventricular wall (Fig. 29). Weight was lent to the validity of this hypothesis by the suggestion that this actually had been accomplished inadvertently in the case illustrated in Fig. 23.

In the implementation of this concept, 7 patients have been operated upon in this manner. While the regurgitation was abolished completely in only 2 instances, it was reduced significantly in all. There was no abnormal tendency to bleeding; there were no operative deaths, and all operated patients subsequently have manifested definite clinical benefit.

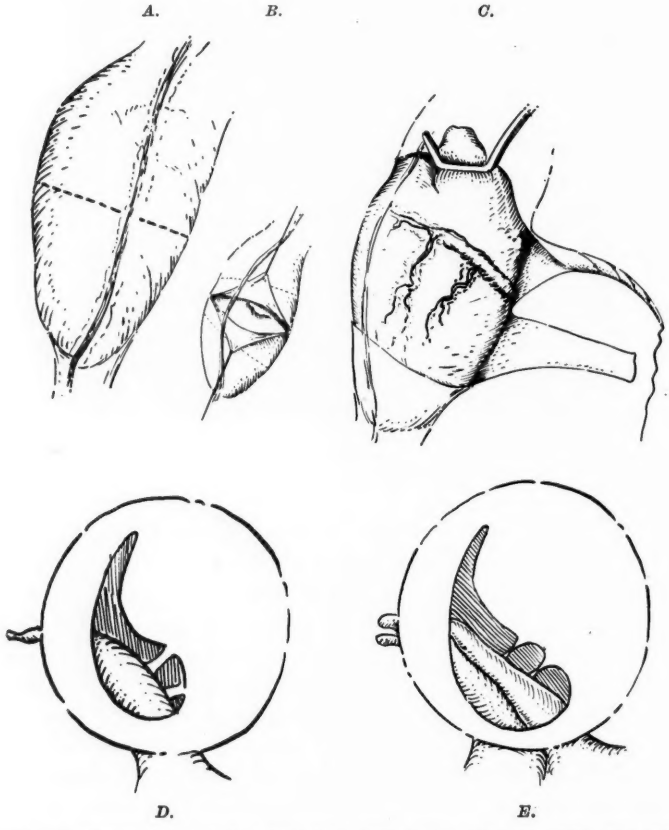


Fig. 29.—*A*, Preparation of two tapering, posteriorly pedicled, pericardial tube grafts. Line of proposed pericardial incision. *B*, Left phrenic nerve is mobilized completely before opening pericardium. *C*, Longitudinal twisting of grafts causes them to assume a tubular character. The epicardial surface must be made to lie on the outside. *D*, Transvalvular graft placed to form a diaphragm of living tissue across the concavity of the relatively or actually deficient mural leaflet. *E*, When a single such graft is not large enough to abolish or nearly abolish the regurgitation, a second one may be placed alongside it (through different myocardial punctures) to widen the restraining diaphragm.

LEFT VENTRICULAR BY-PASS DURING SURGERY

This added ease and facility of intracardiac manipulation has been matched by another important development—the availability of a simple and satisfac-

tory method of partial left ventricular by-pass by use of the Gemeinhardt pump (Fig. 30, *A* and *B*). The construction of this pump and its experimental use and testing were accomplished by two of us (W. G. and W. L. J.). Its specific clinical use has been reported in detail elsewhere.[19, 20]

This pump is connected to the circulation by two cannulae, one inserted by way of a left superior pulmonary vein puncture into the left atrial chamber, while the other is inserted into the aorta by way of an incision into the left subclavian artery. While it is necessary initially to fill the pump with 150 c.c. of saline or blood, and while heparin in a dosage of 1.2 mg. per kilogram is necessary to inhibit coagulation, these quantities are much less than are required for any other extracorporeal circulatory apparatus of which we are aware.

The Gemeinhardt pump may be used in any patient with borderline circulatory competence to assure an effective output of oxygenated blood into the aorta at a pressure sufficient to maintain coronary and generally bodily needs. Thus it is rendered possible to perform manipulations within the heart with the aid of this pump which could not be tolerated otherwise. Dodrill[10, 21] has reported the implementation of a similar concept with a pump of different design. It is believed that many others will adopt this principle in the future as progressively more difficult types of cardiac surgery are attempted in borderline cases in which the patient's circulatory reserve may be insufficient to carry him successfully through the operation without auxiliary support.

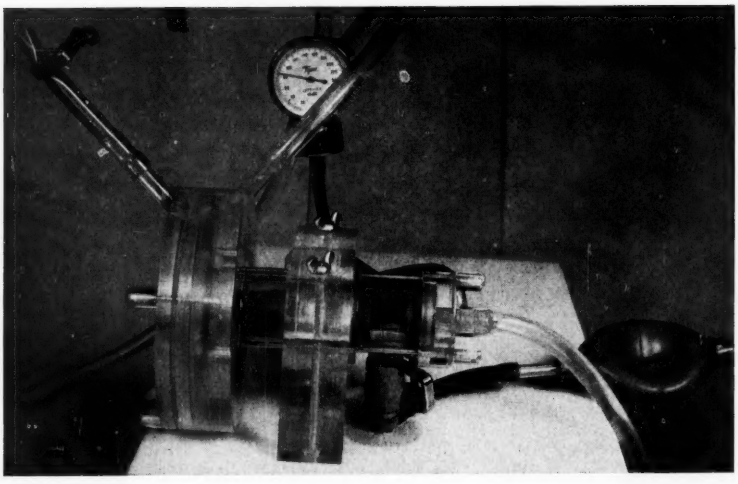
With the advantages of the availability of the mitral stitcher and of the Gemeinhardt pump, we have been able to expand our surgical program significantly to include cases which were previously considered inoperable or at least incorrectible.

PRESENT SURGICAL TECHNIQUE

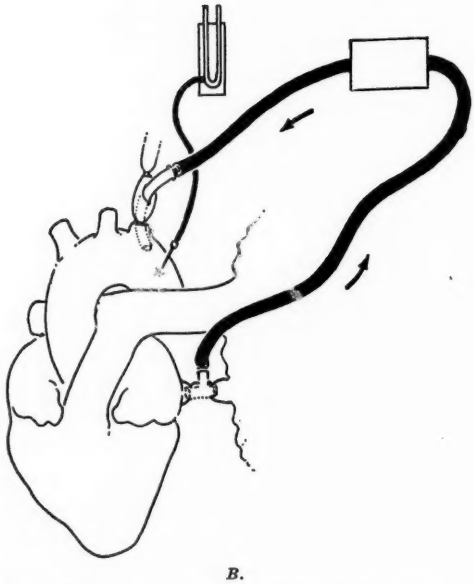
The patient is studied and prepared in the same manner as other cases for mitral surgery. Since 71.3 per cent of these patients have chronic atrial fibrillation, nearly all will be on full digitalis maintenance. After compensation has been established and clinical improvement has reached a maximum, the patient is brought to the operating room and is anesthetized, preferably in the usual manner. This is the combination program of intravenous Pentothal and procaine with endotracheal oxygen which has been described by Keown, Grove, and Ruth.[22]

The patient is placed in the true lateral position with the left side up. The usual curved posterolateral incision is made beginning in the midclavicular line below the breast, and extending below the lower angle of the scapula and up posteriorly to the level of the spine of the scapula. The muscles of the chest wall are divided in the line of the skin incision, and all bleeding points are clamped and ligated or coagulated with the high frequency current.

The fourth intercostal space is opened from the internal mammary vessels to the transverse processes of the vertebrae. If no pleural adhesions exist,



A.



B.

Fig. 30.—Gemeinhardt pump. *A*, Essential parts of the pump. It is effectuated by compressed air from a compressor situated beneath table top. *B*, Diagram showing proposed connections of pump to circulation.

the lung is compressed posteriorly. If there is pleural symphysis, the upper lobe is completely mobilized, but the lower is permitted to remain adherent.

The pericardial sac is opened. The surface of the usually dilated atrium is palpated to detect the pathognomonic systolic thrill of mitral insufficiency. Occasionally, it is absent even when a significant degree of regurgitation exists. When free sutures of pericardium are to be used, the sac is incised longitudinally. If a transvalvular pedicled diaphragm is to be applied, the sac is incised transversely after mobilizing the phrenic nerve. Since this frequently cannot be determined in advance, it is suggested that a very limited pericardial opening be made at first. Then, regardless of what is determined after intracardiac exploration, an advantageous extension of the pericardial opening will be feasible.

USE OF THE GEMEINHARDT PUMP

At this point the patient's condition and tolerance to surgery is evaluated. If it seems probable that his blood pressure and other vital phenomona will be well maintained without the aid of a partial by-pass pump, the definitive procedure is begun cautiously without it. If the blood pressure has dropped significantly or if there are other evidences of circulatory inadequacy, the pump is attached quickly to the vascular system by inserting a cannula into the left atrial chamber by way of a puncture in the anterior surface of the left superior pulmonary vein. The branches of the vein are encircled twice distally with a heavy silk ligature which is tied only moderately tightly. A similar ligature is placed about the proximal portion of the vein to secure the cannula in place as well as to prevent leakage about it. Heparin, 1.2 mg. per kilogram of body weight, is administered intravenously, and blood is permitted to enter the apparatus to fill it up to the filter, and to expel any inadvertently trapped air bubbles.

The left subclavian artery is mobilized to its origin. The proximal portion of the arterial wall is encircled with a heavy silk purse-string suture which picks up the adventitia. The distal portion of the artery is ligated with umbilical tape which is not tied sufficiently tightly to injure the intima. The proximal portion of the vessel is obstructed with a pair of toothed Potts ductus forceps, and a longitudinal incision is made in its exposed surface. The lumen is flushed with dilute heparin solution and the arterial cannula is inserted into its lumen (directed proximally) as the clamp is removed, until the beveled end of the cannula lies within the aortic lumen. The circumferential ligature placed about the proximal portion of the vessel is tied moderately tightly to restrain bleeding and hold the cannula in place.

Arterial blood is admitted to the pump expelling any entrapped air. Now the pump mechanism is set into action and its speed is increased gradually.

A direct mean manometric reading may be obtained from any conveniently located peripheral artery, even from the aorta, by direct needle puncture and the use of a mercury manometer. Alterations in the rate of pumping, and the use of any vasopressive agent are conditioned to the level of the mean blood pressure. This usually should be kept within a range of 90 to 120 mm. mercury.

DEFINITIVE SURGERY OF THE VALVE

Whether the pump is to be used or not, the distal portion of the usually greatly dilated left auricular appendage is encircled with a purse-string suture. This is incorporated within a Rumel-Belmont tourniquet. The appendage is clamped off and the terminal end is incised for a distance of 2 cm. Its cavity is flushed. The index finger of the right glove is amputated and the bare finger tip is inserted between the lips of the appendageal opening. As the occluding clamp is removed, the finger is passed well into the left atrium, hemostasis being provided by tension on the purse-string by way of the tourniquet.

The valve is sought immediately, and its structure is explored. The existence and relative amount of the insufficiency is estimated from the size and character of the regurgitant jet. The size of the valve orifice, the presence and type of any coexistent mitral stenosis, the localization of the jet with relation to the valve commissures, and the flexibility of the leaflets must be estimated carefully. The presence of any calcification and its amount and character are determined.

After a thorough exploration of the valve the operator will have some idea of the type and amount of the insufficiency, the necessity and probable success of corrective surgical measures, and the advisability or necessity of performance of a preliminary commissurotomy. If the latter is required, it is performed at once in order to enlarge the valve opening and to improve its functioning. Sometimes a properly performed commissurotomy alone is all that is necessary to restore a considerable degree of valve competence. If this effect is sufficient, no further corrective surgery is indicated.

However, in most instances the insufficiency will persist. Then the operator may gently insert his finger tip into the incompetent portion of the opening to determine whether there is a relative or actual lack of valvular tissue posteriorly or elsewhere. In many cases the cross-sectional area of the finger will add sufficiently to the "substance" of the retracted mural leaflet to restore complete competence without seriously blocking the size of the valve aperture during diastole. This phenomenon is dependent upon the existence of an adequately functioning septal leaflet (see Fig. 26).

In certain cases one leaflet (usually the mural) will be determined to be suspended at a higher level than the other. This is suggestive of unequal tendinous suspension of the two leaflets and indicates the advisability of placement of an "artificial" chordal suspension as previously described. Digital depression of this higher leaflet usually will bring about perfect valve apposition in these cases.

In most cases the leak will be found to be located posteriorly and one of the forms of direct repair by the use of a free or a pedicled pericardial graft will be deemed advisable, if it is over 2 plus in magnitude. Having estimated, with the finger, the amount of the lack of valvular substance, the finger is removed from the heart and the appendage is clamped.

TRANSAURICULAR SUTURING OF THE VALVE

A full length fairly broad strip of the pericardial sac is removed from the region anterior to the left phrenic nerve. Great care is taken to control any bleeding from the remaining cut pericardial edges or from the dissected mediastinal tissues.

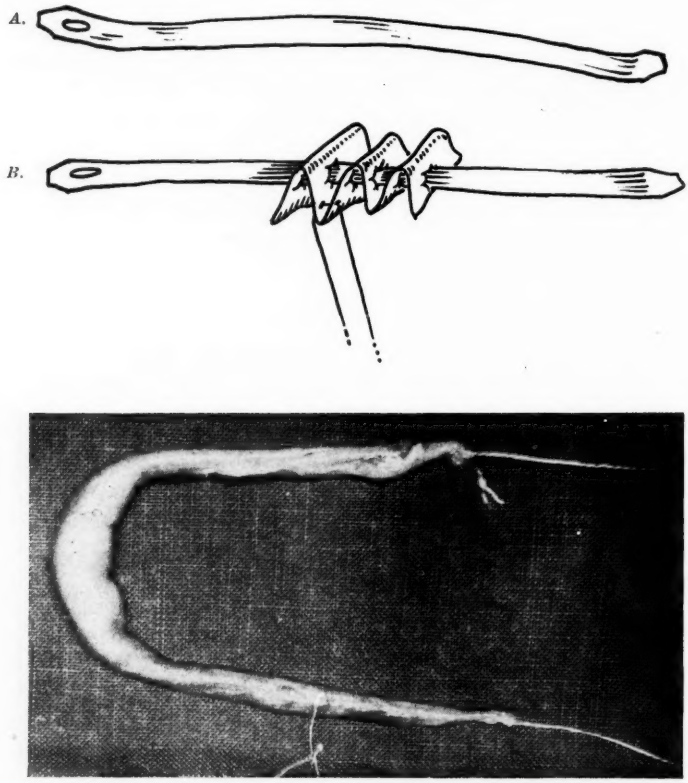
When the valve is of firm structure, the orifice is large, and there appears to be an adequate quantity of valvular tissue for suture approximation without tension, a 3 cm. wide strip of pericardium is obtained to be used as an oblique approximating suture between the portions of the valve edges making up the posterior peripheral segment of the orifice. Both ends of the strip are cut in tapering fashion and an "eye" is created in one end.

When it is felt that, because of an actual or relative lack of valvular tissue, there is a necessity for the application of additional living substance to the posterior incompetent portion of the valve, provision is made for it in either of the following two ways: If the pericardial strip is deemed to be sufficiently long, it is trimmed so that it is 3 cm. wide except in the very middle portion, where all excess pericardial tissue is permitted to remain attached. This is rolled or folded about the central axis of the strip in such a way as to create a mass of pericardial tissue 2 to 3 cm. long, and perhaps as thick as a man's finger. This is to be placed on the ventricular aspect of the valve in its incompetent portion. Since it will swell initially and shrink eventually, considerable care should be devoted to establishing the correct size of the mass or "plug." The selected size is definitely established by the application of securing sutures of fine arterial silk. Both extremities of the pericardial strip are beveled. No "eye" is made.

On the other hand, if the length of the available pericardial ribbon seems short in comparison with the size of the left atrium, it simply may be trimmed to a 3 cm. width with tapering extremities and an "eye" in one end. Then an additional free strip of pericardium may be "impaled" upon the tapered one by passing the latter through each of a longitudinal row of openings made in the former one. This produces an "accordion-pleated" mass which can slide freely upon the length of the tapering strip. (Fig. 31 *A-C.*)

Now, having prepared a suitable "pericardial suture," the bare right index finger is returned to the left atrial chamber. The left-sided "mitral stitcher" is threaded with a heavy nylon suture (No. 2) and is inserted into the heart by passage along the index finger and under the anterior lip of the incised auricular appendage. The open instrument is applied to the free margin of the valve orifice in such a way that the suture-bearing "beak" becomes engaged beneath the mural leaflet. When simple suture obliteration of the posterior incompetent segment of the valve is all that is intended, the beak is placed at a point two-thirds of the distance from the anterior commissure to the posterior (junction of the posterior and middle thirds). When it is intended to apply additional substance to the valve, the site of mural leaflet penetration is selected as that where the leaflet retraction begins to become apparent. This will usually be about at the mid-point in the length of this leaflet.

Having placed the beak properly under the leaflet, the ends of the nylon suture are held under slight tension and the spearlike sliding blade is advanced by pressure of the thumb of the operator's left hand. It pierces the valve substance, creating a 3 mm. long slit, and engages the nylon suture held in the beak within the slotted groove on its inner edge.



C.

Fig. 31.—*A*, Simple pericardial suture. These should be made 3 cm. in width, the full apicobasal length of the pericardium, and should be made to taper at their extremities. *B*, To supply additional graft material, especially with a small graft, a 1 cm. wide additional pericardial strip may be impaled by the original one, through a row of longitudinal punctures made 1.5 cm. apart. This produces an "accordion-pleated" mass which slides freely on the basal suture strip. A simple cotton suture loop should be placed through the substance of the sliding mass to insure against loss into the blood stream in case the nylon sutures become detached from either pericardial end. *C*, Photograph of pericardial suture made with central thickening. No noose may be formed with this type of graft.

By backward traction upon the sliding blade a loop of nylon suture is drawn up into its sheath and finally well out of the heart (at least 8 to 10 cm. out of the instrument). The sliding blade is detached from the suture loop, and the instrument is disengaged from the valve and removed from the heart. One loop of nylon suture and two free ends now emerge from the opening in the

left auricular appendage. The former emerges from the atrial aspect of the mural leaflet of the valve while the latter traverses the natural valve orifice to penetrate the ventricular aspect of the mural leaflet. Each of these three suture ends is fixed *without tension* to the drapes with fine hemostatic clamps.

Now if the posterior peripheral valve segment is merely to be sutured closed, the threaded right-sided "stitcher" is inserted into the heart and a similar nylon suture is placed at the selected point (somewhat more posteriorly than at the junction of the posterior and middle thirds) in the substance of the septal leaflet. The loop and suture ends are brought out of the appendage in fashion the same as the previously placed ones. Such cases will be those with very large valve orifices, and some of them may also require the placement of a second more posteriorly located approximating suture.

When the over-all valve size is less adequate and when one only desires to add extra living tissue to the retracted mural leaflet, the left-sided stitcher is rethreaded and used. This time it is inserted into the left atrium on the palmar surface of the intracardiac finger which is turned in a pronated position, care being taken not to entangle with the previously placed nylon sutures. The "beak" is engaged under the posterior commissural tissue in such a way as to be assured that the graft tissue mass will be well applied to the contracted concave free margin of the mural leaflet. The sliding blade is driven home, perforating the commissural tissue and engaging the suture loop which is then drawn out of the heart. The remainder of the instrument is disengaged from the valve and withdrawn. Again, the loop and two ends of nylon emerge from the appendage and are tacked down without tension to the available wound drapes to avoid possible entanglement and subsequent confusion. (Fig. 32, *A-D.*)

Now the tapering extremities of the pericardial strip each are attached to one of the two pairs of free nylon ends, which pierce opposite valve leaflets (or the mural leaflet and the commissural tissue).

Traction is now cautiously made upon the appropriate sides of the two nylon loops until the free ends attached to the pericardial strip become drawn into the heart. Davidson[23] has aided greatly in the application of this technique by suggesting differential staining of half lengths of each of these nylon sutures.

The traction must never be very forcible, nor must undue tension ever be permitted lest the valve leaflets become "sawed through." Judicious release of the purse-string tension and manipulations with the intracardiac finger aid greatly in the application of the graft to the ventricular aspect of the valve.

If the heart action becomes weak at any time, it probably is related to temporary aggravation of the insufficiency by distortion of the valve by the traction of the nylon or pericardial suture. It can be alleviated by release of the traction and by placement of the finger tip into the incompetent portion of the valve orifice to improve its function until full cardiac vigor is restored. When using the Gemeinhardt pump to maintain the circulation, maintenance of the intrinsic cardiac function is not quite so important during the definitive surgical manipulations since it may be replaced mechanically.

Finally, the pericardial ends having traversed the valve orifice will reach the puncture sites in the valve from the ventricular aspect. They may meet with considerable resistance to further passage because of the smallness of the perforations. However, if the entire graft is inserted into the left atial chamber, to reduce external frictional drag, and if the nylon suture is pulled successively in different directions (cephalad, caudad, anterior, and posterior)

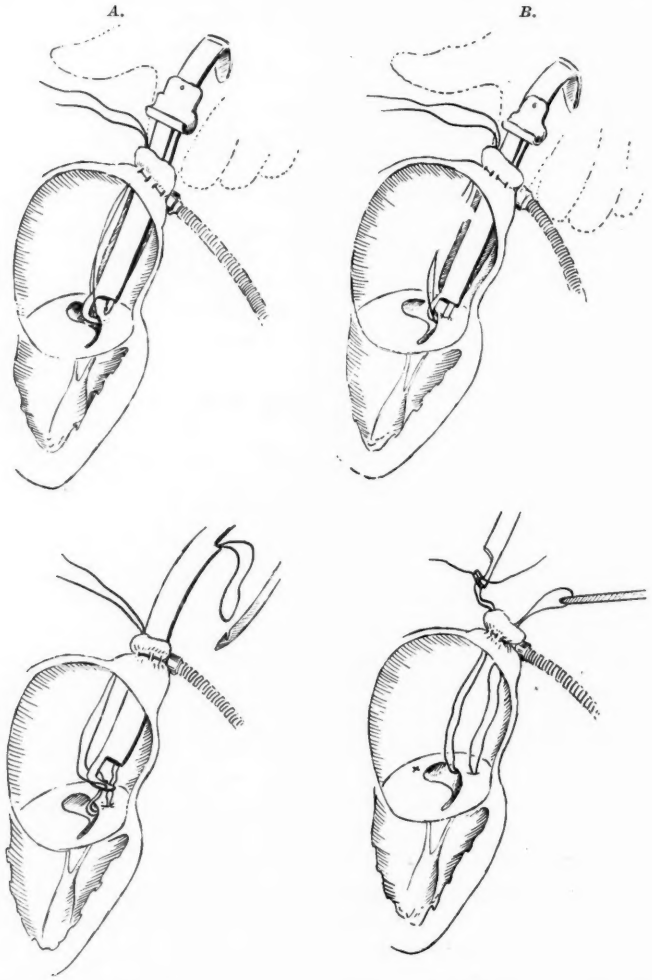


Fig. 32.—Technique of using the mitral stitcher. The stitcher has been inserted into the left atrium alongside the operator's index finger. *A*, Engaging the "beak" threaded with nylon under the free edge of the mural leaflet. *B*, Advancing the sliding blade so that its slotted spear point pierces the leaflet and picks up the suture held in the "beak." *C*, Removal of the sliding blade withdrawing 8 to 10 cm. of the suture loop from the instrument. *D*, After the sliding blade is detached, the rest of the instrument is removed from the heart.

with a release of tension between each change of traction, the tapered graft ends will soon penetrate the punctures in the valve, will emerge from the heart, and the graft will be in place.

If the type and size of the graft have been selected properly, and placement has been correct, the regurgitation should be nearly or completely abolished at once, especially if gentle traction is made upon the nylon ends. This slight tension may be made permanent by passing the intact tapered end of the suture through the previously made "eye" in the other end in the case of the simple approximating pericardial suture and in the case of the suture with an "accordion-pleated" impaled pericardial mass. The fine cotton suture loop, which will have been placed through this "impaled" mass to prevent its possible loss into the blood stream (guard against the possibility that one of the nylon sutures might become detached from one of the pericardial ends) is now cut and removed. When using the pericardial suture with the central rolled pericardial thickening, no noose technique is possible and both pericardial ends must be brought out separately from the appendage. (Fig. 33, *A-F*.)

The mitral insufficiency having been largely or completely corrected, as indicated by the lessening or abolition of the regurgitant jet, and an adequate residual valve orifice having been maintained, the procedure is terminated. The finger is withdrawn from the heart and the appendageal stump is ligated and oversewn. The oversewing sutures are carefully guided through the substance of the emerging pericardial end or ends. No great amount of tension upon them is permitted.

TRANSVALVULAR APPLICATION OF A RESTRAINING PERICARDIAL DIAPHRAGM

Whenever there is any question as to adequacy of the size of the perhaps surgically enlarged valve orifice, when the valve leaflets are thin and presumably fragile, or when a portion of the mural leaflet or the posterior commissural tissue is retracted against the ventricular wall, even oblique placement of a free pericardial suture is contraindicated or technically not feasible. Here a transvalvular pericardial tube diaphragm should be employed.

The operator, after exploring the valve and determining upon the indicated course of action, removes his right index finger from the heart and temporarily clamps the left auricular appendage. He then passes to the opposite side of the table, and, after the patient has been rotated more nearly into the prone position, introduces his ungloved left index finger into the left atrium by way of the appendage. The terminal edge of a specially constructed semimalleable probe* is threaded with heavy nylon thread, care being taken that the latter does not become twisted about the instrument. Guided by the intracardiac finger, the probe tip is inserted through the ventricular wall in a diagonally upward fashion beginning at a point 1 cm. below (toward the apex) the atrioventricular sulcus, over the mid-point of the attachment of the mural leaflet. As the probe tip

*Obtainable from Geo. P. Pilling & Son Co., Philadelphia, Pa., as "transvalvular graft probe."

enters the heart, the intracardiac finger tip guides it close to the free edge of the mural leaflet and across the posterior incompetent portion of the valve orifice to the region of the posterior commissural tissue.

The probe then is caused to penetrate the posterior ventricular wall in a direction diagonally downward (toward the apex), either just above and practically through the annular attachment of the commissural tissue, or, preferably, just to the ventricular side of this attachment.

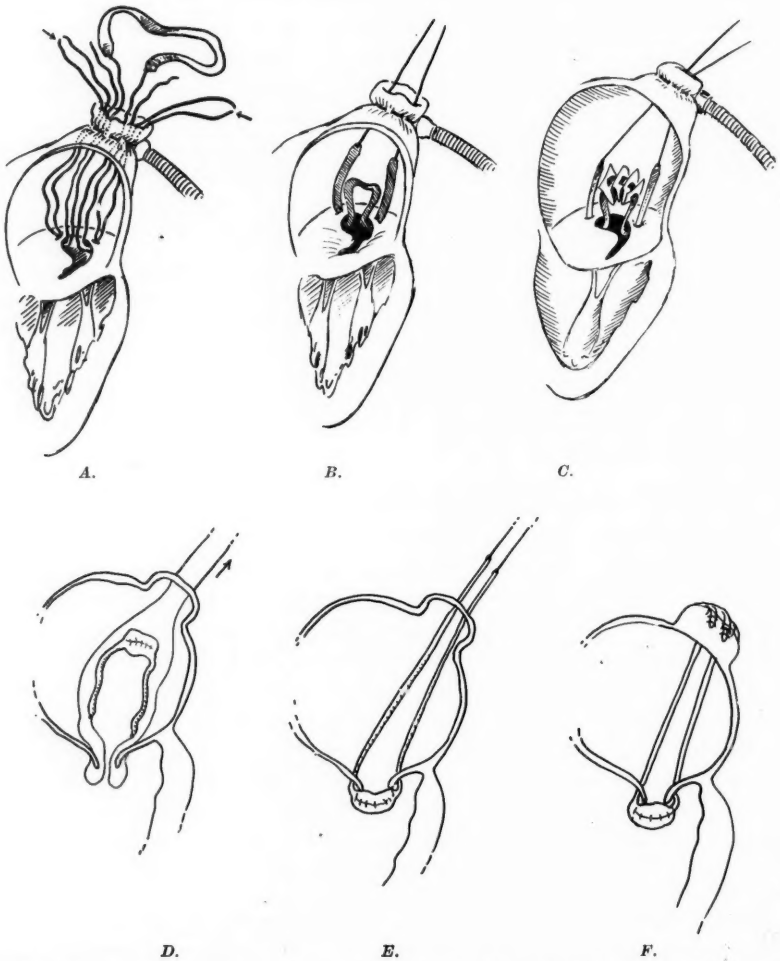


Fig. 33.—*A*, Replacement of the nylon sutures with a pericardial strip. Attachment of pointed extremities of pericardial graft (one of the three types described) to one of each of the central pairs of free nylon sutures. *B*, By traction upon the emerging closed suture loops, the ends of the pericardial strip are caused to enter the heart and to pierce the valve from below. *C*, Impaled pericardial suture piercing the valve. *D*, Pericardial strip with thickened central portion piercing valve leaflets. *E*, Pericardial ends emerging from appendage. *F*, Attachment of free ends of pericardium to appendageal stump.

The probe tip can be felt digitally or can be seen by an assistant, bulging the myocardial tissues before it actually penetrates the epicardium. If it appears about to emerge in close proximity to an important coronary arterial branch, it is withdrawn slightly and redirected to emerge at a more favorable site. Then, the suture borne in the terminal eye is grasped and held, while the probe is drawn back and out of the heart.

Ten or more centimeters of the suture are drawn out of the heart posteriorly and the loop is divided into two separate strands. One of these is set aside as a "spare" in case the other should become detached from the end of the graft. A circumscribing mattress suture of No. 1 braided silk on an Atraumatic R curved needle is placed in the myocardium about the site of posterior suture emergence at a distance of 6 to 10 mm. A single knot throw may be placed to restrain bleeding from this puncture wound. The anterolateral puncture site may be treated in similar fashion.

If this procedure has been chosen initially, the pericardial sac will have been incised transversely (after fully mobilizing the left phrenic nerve) at a level close to the atrioventricular junction posteriorly. Then, two suitable tapering pericardial tubes pedicled posteriorly will have been prepared. If the pericardial entrance has been of any other type, the preparation of even one satisfactory pedicled pericardial tube may prove difficult, but usually can be accomplished. Free grafts are much less desirable because they shrink more. All excessive pericardial fat is removed. In order to obviate the use of sutures, the pedicled tube is shaped by twisting it loosely upon its long axis, taking care that the smoother epicardial surface comes to lie externally. A tube 12 or more centimeters long, prepared from a tapering strip of pericardium 6 cm. wide at its base and smoothly tapering to its tip (not over 1.5 cm. width of pericardial tissue here) is quite satisfactory.

The tip of the graft is attached securely (by suturing) to one of the two strands of nylon emerging from the heart wall posteriorly. The intracardiac finger tip lifts up the transvalvular portion of the sutures to cause the direction of the transmyocardial traction to follow the desired oblique course both anteriorly and posteriorly. Now, the appropriate suture end emerging anterolaterally is drawn upon. The second suture strand acts as a "spare," to be utilized only if the graft should become detached from the first.

The tapering end of the graft penetrates the posterior ventricular wall, traverses the posterior half of the valve orifice, dips under the mid-point of the free edge of the mural leaflet, and emerges from the lateral left ventricular wall. The intracardiac finger determines the point at which a sufficiently large bulk of graft tissue lies within the incompetent portion of the valve orifice. Should weakening of the cardiac activity occur at any time, further manipulation of the probe or graft should be held in abeyance until improvement in function takes place. Placement of the finger tip within the incompetent portion of the valve orifice, as described previously, will tend to bring about improve-

ment by temporarily abolishing the valvular leak. After restoration of cardiac vigor has been accomplished, the definitive reparative manipulations may be resumed cautiously.

The degree of correction of the insufficiency finally achieved may be estimated digitally by evaluating the extent of reduction of the regurgitant jet. The unhampered action of the septal leaflet and the size of the remaining valve aperture should be noted at the same time. If it is determined that another tube diaphragm is necessary to abolish the leak, the procedure is repeated and a second graft is placed alongside the first but through slightly different puncture sites.

The procedure is terminated by tying the posterior mattress suture securely about the graft after removing the second transcardiac nylon ("spare") suture. Usually, we prefer also to attach the mattress suture to the graft substance by direct suturing. It is well to have retained some loose or excess graft tissue posteriorly so that the cardiac movements will not make traction upon the graft and, hence, will not tend to cause bleeding from the site of entrance into the ventricle.

The anterior end of the graft is fastened in a similar fashion, using a mattress suture in the myocardium and affixing it securely to the graft. The end of the latter, if sufficiently long, may be tied into a "knot" which then is rendered a permanent one by placing transfixion sutures of cotton. Such a bulky knot of living pericardium tends still further to seal the ventricular wound against hemorrhage and to insure against any possibility that the end of the graft might be drawn into the heart by the systolic surge of ventricular pressure against the transvalvular portion of the graft diaphragm.

If the Gemeinhardt pump has been used, its action is caused to slow down gradually to permit the left ventricle to take over its full natural burden, now much reduced by the effectiveness of the surgery. The atrium is decannulated by cutting the proximal encircling suture about the pulmonary vein and removing the cannula. The central end of the vein is clamped simultaneously, and it is flushed with heparin solution and repaired with arterial sutures. Then the clamp and the distal encircling sutures are removed restoring normal pulmonary drainage.

The subclavian cannula is removed after cutting the proximal encircling suture. The vessel may be clamped proximally and repaired by suturing, or simply may be ligated centrally and distally.

Chest wall closure is routine, using simple water-seal drainage through a fenestrated No. 26 urethral catheter inserted through a stab wound in the left eighth intercostal space, posterior axillary line.

The correction of insufficiency due to unequal suspension of the leaflets has been detailed (Fig. 35, *A-E*).

The roentgenograms on two patients treated by valvular suturing are shown in Figs. 36 and 37.

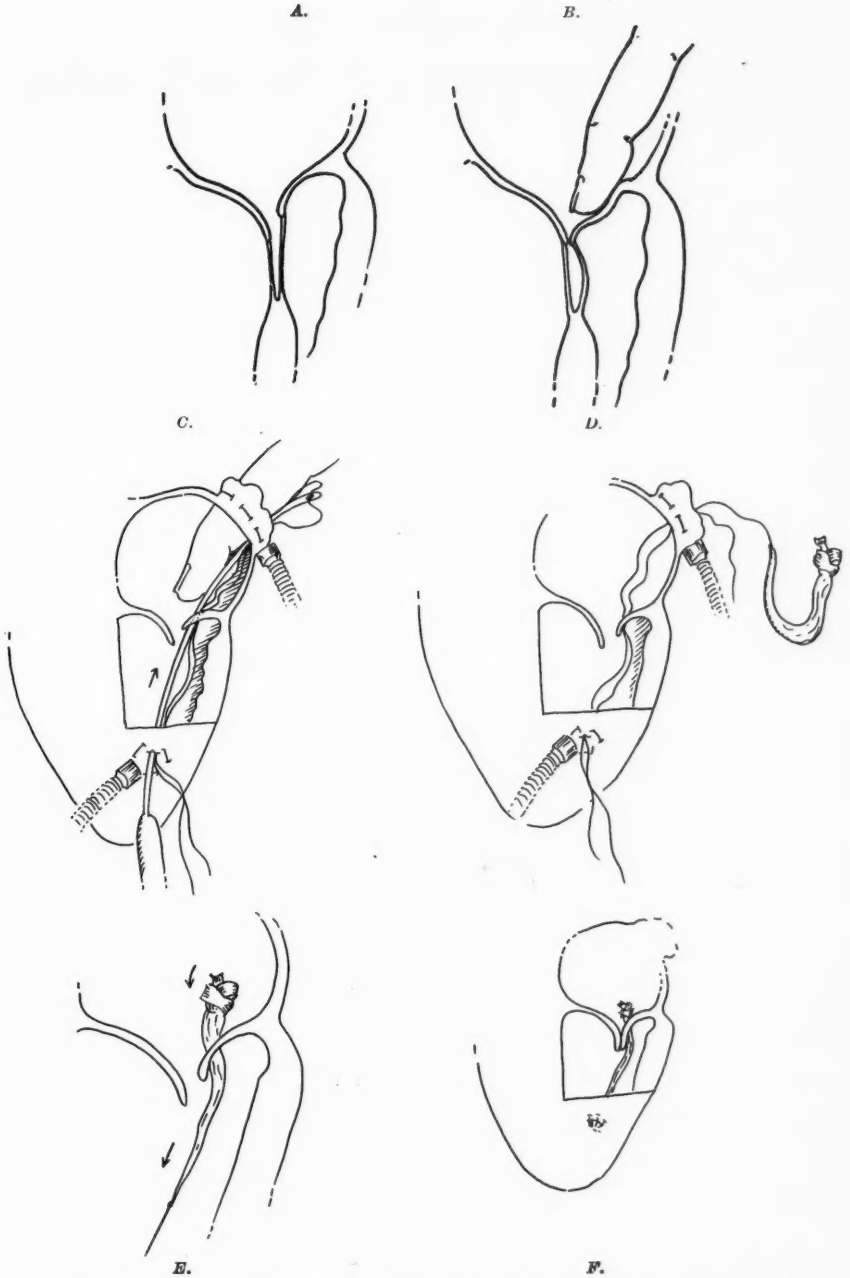


Fig. 34.—Jamison method of correcting mitral insufficiency due to unequal shortening of suspending chordae. *A*, Long section of valve showing unequal levels of suspension. *B*, Temporary restoration of competence by digital depression of the higher leaflet. *C*, By transventricular passage, threaded probe pierces the higher leaflet and follows the finger to emerge from left auricular appendage. *D*, Suture is grasped, probe is withdrawn, and the atrial end of the suture is attached to the tapering end of a knotted pericardial graft. *E*, Pericardial strip drawn into heart. *F*, End of new support secured to epicardium.

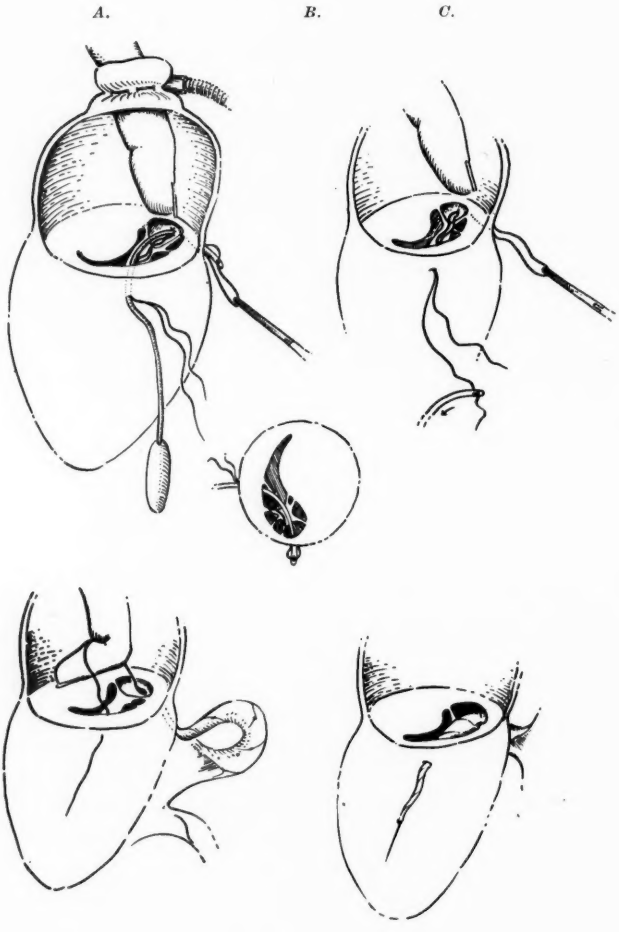
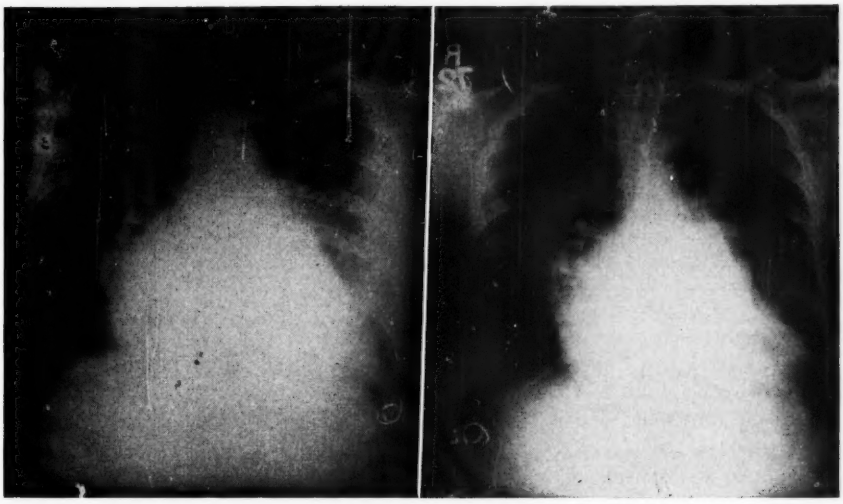
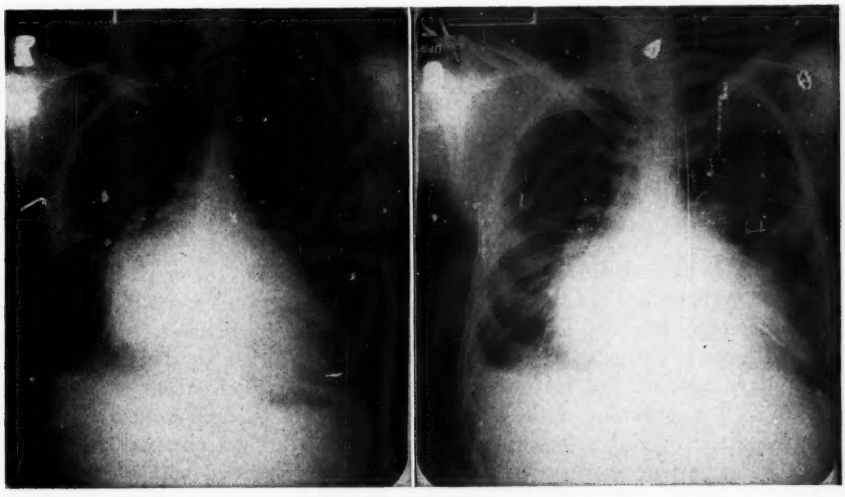


Fig. 35.—Steps of placement of transvalvular pericardial diaphragm. *A*, Passage of curved terminally threaded semimalleable probe, guided by the intracardiac left index finger, through left ventricular wall just beneath middle of mural leaflet, diagonally across the posterior portion of the valve orifice, and finally through the ventricular wall in the region of the posterior commissural tissue. *B*, Top view of course of passage of the probe. *C*, Suture borne in tip of emerging probe is grasped and held by an assistant. Probe is then withdrawn. *D*, After dividing posterior suture loop, one end is secured to the tip of the graft; the other is retained as a "spare." The intracardiac finger lifts up the transvalvular suture loop in order to direct the transmyocardial traction along the same lines as the course of the probe punctures. *E*, By traction upon the anterior extremity of the suture, the graft is drawn through the heart and across the valve orifice until the operator, guided by his intracardiac finger, is satisfied as to the degree of reduction in the regurgitation. If not, a second passage of the probe may be required for the placement of a second parallel graft.



A. *B.*

Fig. 36.—*A*, Preoperative roentgenogram in S. A., a 19-year-old white man with wide open (4-plus) mitral insufficiency due to annular dilatation. Almost armchair life. Loud, long apical systolic murmur. Treated by suture obliteration of posterior peripheral segment of valve orifice with two simple pericardial sutures. Regurgitation deemed reduced 75 per cent. *B*, Six months' postoperative roentgenogram showing significant reduction in heart size. Clinical improvement has been extremely satisfactory. Much reduced systolic murmur.



A. *B.*

Fig. 37.—*A*, Preoperative roentgenogram in C. D., a nearly completely incapacitated 24-year-old white man with severe predominant mitral insufficiency (3-plus) but some significant stenosis. After anterior commissurotomy and posterior commissurorrhaphy using a large (6 cm. wide) suture to add pericardial substance to the free posterior margin of the mural leaflet, the regurgitant jet was completely abolished. *B*, Three months' postoperative roentgenogram shows no apparent reduction in heart size. Nevertheless, there is now only a 1-plus systolic murmur and patient can climb three flights of stairs without difficulty.

The hospital mortality experienced in the treatment of 156 patients with severe mitral insufficiency is presented in Table V.

TABLE V. METHODS OF MANAGEMENT IN 156 PATIENTS OPERATED UPON FOR MITRAL INSUFFICIENCY

	NO. OF CASES	DIED	MORTALITY (%)
I Transventricular pericardial hammock	52	20	38.5
II Transventricular suturing of the valve	72	20	20.8
III Modern valvular suturing	25	4	16
IV Transvalvular posterior pericardial diaphragm	7	0	0
Total	156	44	28.2

SUMMARY

1. Mitral insufficiency in well-developed form is one of the two most devastating acquired valve lesions.

2. A functional and a pathologic classification of this condition has been presented.

3. An anatomical and pathologic explanation of the two commonest types of mitral insufficiency is given. The characteristic tendency toward posterior localization of the incompetence is pointed out and a suggested explanation has been advanced.

4. The ultimate fate of pericardial tissue inserted into the heart is shown.

5. The various types of valvular suturing and their basic underlying principles of action are detailed.

6. The concept and application of a pedicled transvalvular pericardial diaphragm is described.

7. The use of the "mitral stitcher" for valvular suturing by the transatrial approach and the use of the Gemeinhardt pump in supporting and maintaining the circulation in unstable cases are indicated and the technique of their use is described.

8. The results of these corrective attempts to date have been summarized.

CONCLUSIONS

We believe that mitral valvular repair by grafting free or pedicled pericardial tissue within the valve orifice is a completely sound and logical method of permanently relieving mitral insufficiency in most clinical cases.

In certain patients, suspension of one leaflet by an artificially created "papillary support" of pericardial tissue is extremely valuable.

With the evolution of these methods and increased understanding of the problems, the operative mortality and the clinical results obtained will soon become comparable in mitral insufficiency to those achieved with other forms of mitral valve surgery.

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(*For Discussion, see page 624.*)

THE SURGICAL CORRECTION OF MITRAL INSUFFICIENCY

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INTRODUCTION

IMPROVED techniques for the correction of mitral stenosis by valvuloplasty have brought the problem of mitral insufficiency into sharper focus. If a reasonable surgical treatment for this lesion can be offered, the whole field of mitral valve surgery will be improved in three ways. Those suffering from pure mitral incompetence can be treated. Those who might have either stenosis or insufficiency, now being denied surgery because of this question, can be operated upon definitively. Finally, those individuals thought to have mitral stenosis who at operation prove to have free insufficiency can be treated rather than abandoned.

During the past six years, in the course of treating more than one thousand patients with diseased mitral valves, there has been a unique opportunity to gain firsthand information about the nature of mitral incompetence: clinically, pathologically, and through observation and palpation at the operating table. While not within the province of this presentation, the life cycle of mitral insufficiency has become clarified, the diagnosis and recognition have been improved, and, therefore, the indications for surgical intervention are better defined.[1, 2] Simultaneously, a better understanding of the morphologic pathology has been translated into a working concept of *kinetic pathology*, i.e., the total effect on hemodynamics in the living state of the diseased leaflets, myocardium, annulus, chordae tendineae, and papillary muscles.

The purpose of this communication is, first, to outline the patterns of morphologic pathology and to describe the kinetic pathology of this lesion as observed in the living human being. With this foundation an appreciation of the cardinal importance of the preservation of leaflet mobility becomes apparent. The fundamental attributes of pure mitral insufficiency are clari-

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Read at the Thirty-fourth Annual Meeting of The American Association for Thoracic Surgery at Montreal, Quebec, May 3-5, 1954.

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fied. It will be shown that in pure or mixed mitral insufficiency one or a combination of conditions must exist: (a) the leaflets may be too short, (b) the annulus may be too large, (c) the leaflet moorings may be defective, or (d) leaflet margins may be of such texture or configuration that they close incompletely. It will be demonstrated that the problem in pure mitral insufficiency is that of the *absolute* or *relative loss of leaflet substance*. To correct this loss of substance there must be either a substitution of substance or reduction of the size of the annulus. Various techniques that have been advanced to accomplish these ends will be reviewed. Evidence is presented in support of the use of a smooth Lucite baffle or prosthesis, so placed in the mitral orifice as to obstruct the regurgitant jet in systole without interfering with leaflet mobility and the forward blood flow in diastole.

The operation to be presented represents one that has been evolved in the laboratory and applied to human beings. The first baffle was spherical in shape, the ball-baffle. The second was more effective as it was bottle-shaped and was placed across the orifice with the base anchored to the anterolateral aspect of the annulus with the neck of the bottle riding under the opposite portion of the annulus. A number of problems presented by this prosthesis will be discussed. The spindle-shaped baffle, anchored at both ends across the orifice, is apparently the most stable and efficient. The rationale and technique of this operation are presented for consideration in the light of the early clinical results on seventeen patients.

MORPHOLOGIC PATHOLOGY

In mitral valvular disease a whole spectrum of pathologic states, ranging from pure stenosis to pure insufficiency, exists. Very little is known about the pathogenesis of these lesions, but it would appear reasonable to assume that stenosis occurs when, in the bullous phase of acute rheumatic valvulitis, the edges of the leaflets are forced together and adhere. This fusion probably progresses from the periphery where the range of motion is least and gravitates centrally until significant obstruction occurs. It is also conceivable that broad zones of swollen leaflets pressed into apposition can seal together as a relatively acute process. Such a sequence of events would result in mitral stenosis.

It is not difficult to conceive of a less intense inflammatory reaction or even a different type of reaction resulting in scarring, shortening, and contracture of leaflets and chordae tendineae without causing them to adhere. This would produce an incompetent valve. In our experience the end result of such a pathologic process can be classified in five general patterns.

Type I. Absolute Loss of Substance.—This is the basic form mentioned previously wherein the rheumatic process has attacked the leaflets directly and the resulting contracture prevents them from closing in systole (Fig. 1, *I*). Because of its important function in the left ventricular outflow tract, damage of the aortic leaflet results in the most serious incompetence.

Type II. Relative Loss of Substance.—This occurs either as the result of rheumatic carditis which attacks the annulus directly or because of generalized left ventricular dilatation secondary to myocardial disease and failure. As the annulus enlarges, the valve leaflets, otherwise adequate in length, fail to approximate, and regurgitation results. This mechanism includes those forms of incompetence often referred to as "functional insufficiency" (Fig. 1, *II*).

In our experience almost all *pure mitral insufficiency* results from a combination of the afore-mentioned two mechanisms. Conspicuous features of pure insufficiency are the absence of calcification and the resultant free

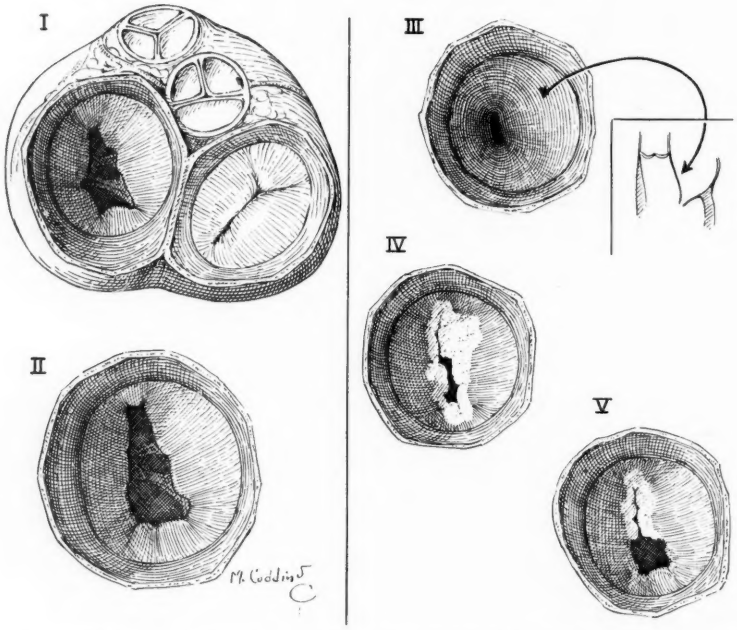

Fig. 1.—The five basic morphologic types of incompetent mitral valves. *I*, absolute loss of substance; *II*, relative loss of substance; *III*, directional; *IV*, malocclusion; *V*, mixed pathology.

mobility of the leaflets. With mitral incompetence resulting from absolute loss of leaflet substance, left ventricular chamber dilatation must occur to compensate for the volume regurgitated with each systole if cardiac output is to be maintained. This in turn produces enlargement of the valve ring with relative loss of substance augmenting the regurgitation. Thus the peculiarly self-aggravating nature of this disease becomes apparent.

In addition to these "pure forms" of mitral regurgitation, there are three other forms in which the predominant problem is usually stenosis. In general, our policy has been to treat only the stenosis. Since there is some incompe-

tence of the mitral closing mechanism, however, these pathologic processes must be included in a morphologic classification of mitral insufficiency.

Type III. Directional.—This form of mitral insufficiency occurs when a stenotic mitral orifice faces into the left ventricular outflow tract (Fig. 1, *III*). The nature of the abnormally rigid stenotic orifice prevents closure, and its position precludes obstruction by the ventricular wall in systole. This type of valve has been described previously as Group B mitral stenosis.[3, 4]

Type IV. Malocclusion.—The regurgitation in this instance results from the inability of irregularly calcified leaflet margins to seat properly when the valve closes (Fig. 1, *IV*). The result is a ribbon-shaped jet. Again, since mitral stenosis is the predominant lesion in most of these cases, correction aims only at relief of the obstruction.

Type V. Combined Pathology.—Finally, a significant number of cases have been encountered in which fusion of leaflet margins and heavy calcification (the pathology common in pure mitral stenosis) will be found at the anterior commissure, while posteromedially there is loss of leaflet substance without calcification, resulting in a significant area of insufficiency (Fig. 1, *V*).

The quantitation of stenosis and insufficiency in such instances, and thus the management of such a lesion, presents a difficult problem. We have had numerous brilliant clinical results following the correction of stenosis alone when this type was found even though there is substantial associated incompetence. When the degree of stenosis is negligible, however, treatment must be directed at the dominant insufficiency. A discussion of how to determine whether stenosis or regurgitation is dominant in such a situation properly falls within the province of kinetic pathology or hemodynamics, but in order to emphasize the correlation of the morbid pattern with the dynamic valve function, it may be said that the identification of the significant lesion can often be made at the operating table. This is done by using the intra-auricular finger as a "baffle" or substitute for the lost leaflet substance; the regurgitant jet can thus be obstructed without interfering with leaflet mobility. Ventricular filling proceeds normally as the leaflets fall away from the finger, but the leaflets plus the finger prevent regurgitation in systole. If the leak is significant, the blood pressure will rise from 20 to 50 mm. Hg within a few beats.

KINETIC PATHOLOGY OR HEMODYNAMICS OF MITRAL INSUFFICIENCY

There are at least three features that should be reviewed.

1. The first of these involves the pressure relationships between the left auricle, the left ventricle, and the aorta. It is at once apparent that the gradient *in systole* between the left ventricle and the left auricle is much greater than the gradient between the left ventricle and aorta. Thus, if the orifices were of similar size, the incompetent mitral opening would allow much more blood to regurgitate through it than would be ejected into the aorta as effective cardiac output. Nevertheless, in the face of this disadvantageous pressure relationship favoring loss of effective systolic ejection from the left ventricle, a quantity of blood equal to the combined aortic output and the

regurgitant jet must flow from the auricle into the ventricle in each diastolic period. That is, auriculoventricular flow in diastole must equal the combined volume of blood put out into the aorta and back into the auricle during each contraction (Fig. 2, upper pair). In addition, there is another deterrent to maintaining adequate ventricular filling in diastole. The forward flow in diastole is at a low pressure gradient and therefore at a low velocity, whereas the reflux in systole is at a high gradient and velocity. There are two principal mechanisms of compensating for this unfavorable dynamic situation. One

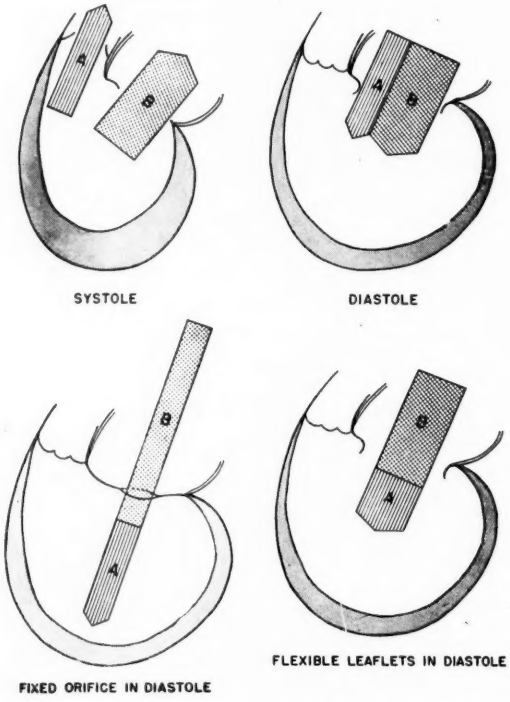

Fig. 2.—(Upper pair) The combined volume of *A* and *B* must flow into the ventricle during each diastolic period. The pressure gradients are unfavorable to the forward flow as opposed to the rapid systolic reflux.

(Lower pair) The two principal compensating factors for this larger volume of forward flow at a lower velocity are prolongation of diastole and increase in the size of the mitral orifice (largely through leaflet mobility).

is that the slower flow from auricle to ventricle may exist over a longer period of time, i.e., *diastole may be prolonged.* The limitations of bradycardia are apparent although a given degree of incompetence will obviously be better tolerated with a slow rate. The principal mechanism by which circulation is maintained is therefore through change in the size of the mitral orifice so that the large volume of blood traveling at a low velocity in diastole proceeds through a larger orifice than the regurgitant jet in systole. This depends on

the maintenance of leaflet mobility (Fig. 2, lower pair). The partial occlusion of the orifice by the ventricle or by annulus contractility are probably additional minor factors. The cornerstone of our efforts at surgical correction of this lesion has been this important preservation of leaflet mobility so that the orifice can be larger in diastole and can become smaller in systole.

2. The second important concept of the kinetic pathology of this condition is that mitral regurgitation is a peculiarly self-aggravating disease. When a significant amount of regurgitation occurs, a certain percentage of the left ventricular stroke volume is dissipated in retrograde flow. In order to maintain an adequate cardiac output, the left ventricle must dilate to accept a compensating volume of blood. This leads to an increase in the size of the annulus which in turn increases the *relative insufficiency* of the leaflets. More regurgitation leads to more dilatation, and a vicious cycle is established. Thus, the self-aggravating nature of this condition is defined. Conversely, any reduction of reflux reduces ventricular volume, and as the mitral annulus shrinks simultaneously, the leaflets more nearly approximate. Effective partial correction of this lesion should therefore be *self-perpetuating,* provided that the ventricular myocardium has not exceeded the limits of its recuperative powers. Failure to make adequate allowance for this dynamic change may result in the creation of stenosis by a procedure which attempts to correct the leak totally at the time of operation. Our experience in this regard will be described.

3. The third cardinal concept has already been mentioned. When the surgeon encounters free or significant mitral insufficiency on exploring the mitral valve, it is possible to hold the finger in the regurgitant jet at the leaflet level in such a way that the leaflets slap firmly against it, and a more competent valve results. This is uniformly associated with a prompt elevation of the blood pressure by as much as 50 mm. Hg. Although instantaneous cardiac output determinations are not available, this prompt blood pressure elevation must correlate with an elevated cardiac output. The reduction of the regurgitation must also reduce the left ventricular work. It is felt that these salutary changes can be simulated on a permanent basis by the surgical insertion of baffles.

SURGICAL CORRECTION

Considerable ingenuity has been demonstrated in devising surgical procedures for the correction of mitral insufficiency. Murray, Wilkinson, and McKenzie[5] in 1938 tried experimentally and then in man, to resect a portion of the mitral valve, replacing it with a tissue sling. After turning a segment of the cephalic vein inside out, they pulled a strip of tendon through its lumen. This sling was then suspended between the anterior and posterior walls of the heart with the hope that in systole it would be blown into the valve defect. They reported that the method has been used in thirty-seven human patients with a 20 per cent mortality. Fifty-five per cent of the patients were reported to have been improved, and 25 per cent were classified as unsatisfactory late results.

Bailey and his colleagues[6] repeated this operation in animals and human beings suffering from mitral regurgitation employing a sling made from a pericardial pedicle graft. Adequate experimental and clinical trial has now established the fact that these "slings" do not survive but shrink to atrophic fibrous bands. Among those who have had similar experiences are Glover and his associates,[7] and Moore and Shumacker.[8]

Certain objections have existed which have led us to doubt the validity of this approach. First, the slings do not survive, as already mentioned. Second, whether they lived or atrophied, they offered a framework for thrombus formation with the threat of embolization or overt obstruction. Third, even though the sling continued to function as planned, there existed the strong probability that it would become jammed into the orifice with the force of ventricular systole in such a way that the much feebler force of diastolic flow could not dislodge it. Finally, an understanding of the kinetic pathology outlined previously indicated that if a sling of the proper length to correct a given state of insufficiency in a ventricle and annulus of one size did in fact correct that regurgitation, the size of the ventricle and annulus would diminish so as to render the sling no longer suitable. (This particular pitfall in our own work will be presented.) For these and other reasons, the procedure has been abandoned even by its early proponents.

Years ago when the valvuloplastic techniques for mitral stenosis were undergoing development it seemed possible that mobilization of the leaflets might improve the hemodynamics of insufficiency. The limited place for such an approach is apparent from the discussion previously of the morbid pathology of regurgitation. Similarly, the limited application of "unrolling leaflets and stretching chordae" as described by Dodrill[9] is apparent. We have shared the optimism and frustration of others in pursuance of these proposals.

Recently Davila, Mattson, O'Neill, and Glover[10] have described an ingenious experimental technique of reducing the size of the annulus. Previously we have repeatedly observed that pressure on the posterolateral aspect of the left ventricle can distort the annulus of an incompetent mitral valve into an elongated elipse so that the absolutely or relatively inadequate leaflets are rendered competent. This observation prompted us on three occasions to place a distorting suture obliquely across the annulus from the posteromedial to the anterolateral aspect; the ends were anchored externally on nylon buttons. One of the patients succumbed soon after operation, and it was found that the degree of distortion possible had been quite insignificant due to sheer muscle mass of the hypertrophied myocardium (Fig. 3). A second patient survived, and there was evidence both at operation and subsequently of improved hemodynamics and reduced regurgitation. Unfortunately, she had a fatal cerebral embolus six weeks later. A third patient has been followed for more than a year. In this instance the palpable regurgitation was completely obliterated by the annulus distortion at the time of operation, but a systolic murmur and presumably some regurgitation have persisted. While the patient may be regarded as improved, the operation has not

constituted definitive therapy. Because of the difficulty in significantly altering markedly hypertrophied ventricles, because of the inherent danger of embolus associated with open transorificial sutures, and because of the possible variation in size in different phases of the cardiac cycle, this technique has now been abandoned.

On reviewing the afore-mentioned experimental and clinical work, it would appear that the use of living tissue is unsatisfactory. At the same time we have been reluctant to adopt nonliving prosthetic valves with moving parts for fear they would wear out or stick. The paradox then presents itself of

Fig. 3.—Autopsy specimen. Hypertrophy of the myocardium rendered distortion of this annulus difficult. The incompetence was not corrected by the transorificial suture. Note external buttons anchoring ends of suture.

being unable to accept living tissue and nonliving substance with moving parts. A compromise in this dilemma appears to exist in a nonmoving, nonviable prosthesis that may be moved by or with the adjacent myocardium and at the same time preserve the vital mobility of the leaflets.

THE EVOLUTION OF THE SPINDLE-BAFFLE OPERATION

Our first attempt to employ a prosthesis to supplement valve substance in mitral insufficiency was with the Lucite ball-baffle procedure. The plan was to mount the ball on the ventricular myocardium just beneath the ventricular leaflet. This ball was not to move on its moorings but was *to be moved* by the myocardium into the deficient mitral orifice during systole and to fall away from the opening in diastole (Fig. 4). The Lucite sphere was introduced through the auricular appendage and drawn into position by sutures that had been inserted through the ventricular myocardium. The

maneuvers were essentially similar to those used and illustrated here in connection with the spindle-baffle operation. The results of this operation were discouraging. The eight patients on whom it was performed died in from one day to nine months after surgery. It was apparent that the terminal course in these dying patients had not been interrupted. Examination of the prostheses at autopsy confirmed the laboratory evidence that they were well-tolerated and did not erode the myocardium. Analysis of these human cases, however, revealed a fallacy in our concept of the shape of the left ventricle

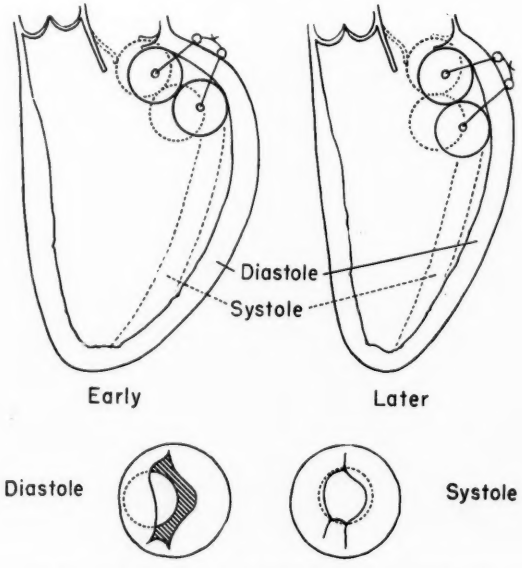

Fig. 4.—This theoretical action of the ball-baffle was not accomplished because of the abnormal contour of the dilated ventricle in mitral insufficiency.

in mitral insufficiency. We had failed to take into consideration the enormous dilatation of this chamber that was present. Consequently, when the ball was placed on the myocardium beneath the ventricular leaflet, it, in fact, came to lie lateral to the valve orifice and not near the orifice where the motion of the myocardium could bring about the desired occlusive effect in systole. The ball simply had no effect on the orifice in life even though in the atonic state at autopsy the position seemed satisfactory (Fig. 5).

The failure of the ball-baffle led to an alteration in its shape so that it could traverse the incompetent orifice effectively. A Lucite prosthesis in the shape of a bottle was anchored above and below the anterolateral aspect of the annulus, and its neck engaged either under the midportion of the aortic leaflet or under the posteromedial fringe of leaflet insertion on the annulus. By placing the baffle properly in the mitral defect, the same elevation of systolic blood pressure brought about with the finger could be duplicated

(Fig. 6). Twenty-four terminal patients with pure mitral insufficiency were operated on using this procedure. There were seven postoperative and seven late deaths. An analysis of these deaths in terms of principal cause is relatively simple, but in actual fact, except for four technical difficulties incident to the insertion of the prosthesis, the terminal event was generally a combination of more than one factor. In three patients the prosthesis that seemed appropriate in size to correct the defect encountered at operation did eventually overcorrect the insufficiency when the chamber contracted and reduced the annulus in size. The reversal of the "self-aggravating cycle" described earlier was effected with fatal consequence. Five patients succumbed from emboli apparently propagated from exposed suture material. There was one embolus of the prosthesis itself. This bottle had been inadequately anchored due to technical problems at operation. It represents the only such tragedy in the series. Three of the patients who succumbed from emboli and two

Fig. 5.—Autopsy specimen. Ball-baffle appears to lie in effective position beneath ventricular leaflet on left side of illustrations. Note Type I and Type II pathology. Tricuspid valve is at right.

others who have succumbed had persistence of irreversible congestive failure. Of the ten surviving patients, there has been substantial improvement in eight, even with rehabilitation to a normal level of activity from a preoperative status of invalidism.

The inconstancy of improvement from patient to patient with the bottle-shaped prosthesis was felt to be correlated with inconstancy of the position maintained by the baffle. Technical difficulties of baffle insertion attended the initial efforts. Exposed sutures were associated with lethal emboli. Overcorrection of regurgitation with resultant stenosis also made any benefit from the operation difficult to discern. Finally, the patients themselves were in most instances accepted sufficiently late in their disintegrating phase that

even a perfect mechanical valve might go unrecognized in the face of malignant myocardial, pulmonary, and liver changes (Fig. 15). However, the incontrovertible improvement of some of the patients confirmed the correctness of the principles involved.

The apparent virtues of the method demanded that: (1) the technique of insertion be made simple and readily reproducible; (2) the baffle be inserted in an effective position with both ends anchored so that the variations in clinical results attending alteration of the baffle position do not occur; (3) that exposed suture material be eliminated if propagation of clot and therefore embolization are to be avoided; and (4) a baffle of just sufficient size to initiate the self-perpetuating return to mitral competence must be inserted so that mitral stenosis does not occur.

The first or *technical* difficulty seems to have been overcome in the so-called spindle-baffle procedure illustrated here. The operation has been carried out seventeen times with three surgical deaths and one late death. None of the deaths were due to technical problems at operation.

The second problem, that of *anchoring both ends of the baffle* so that the defect inherent in the bottle procedure—the possibility of the unanchored end becoming dislodged—has been solved. The anterior and narrower end is anchored under the leaflets and annulus within the chamber of the ventricle, whereas the larger end of the teardrop-shaped spindle is firmly fixed above the annulus posteriorly. Thus, while substituting for the loss of substance and allowing the continuation of full leaflet mobility, the baffle cannot migrate out of position (Figs. 7 and 14).

The third problem of *embolization* or propagation of thrombus from exposed sutures has apparently been controlled. There have been none thus far. This improvement is almost certainly related to the fact that the technique illustrated allows firm anchoring with minimal exposed suture material. The patients have been placed on Heparin immediately after surgery for five days, then shifted to Dicumarol for five weeks.

The fourth pitfall—that of inserting too large a baffle and eventually *overcorrecting to mitral stenosis*—has not occurred since the spindle-baffle has been used. Recognition of the potential problem is the best prophylaxis in this situation.

THE TECHNIQUE OF THE SPINDLE-BAFFLE OPERATION FOR THE CORRECTION OF MITRAL INSUFFICIENCY

The patient is anesthetized with Pentothal and an endotracheal tube is inserted. Minimal amounts of ether maintain anesthesia. The respiratory assistor[11, 12] is used routinely, thus ensuring maximal oxygenation and carbon dioxide clearance.

With the patient in the right lateral decubitus position the appropriate skin preparation and draping are followed by an incision over the entire left sixth intercostal space. The interspace is opened and the sixth rib is divided at the transverse process posteriorly and through the cartilage at the sternum.

If necessary, the fifth costal cartilage is also divided. With the ribs spread the classical cardiac and pericardial silhouette presents. The pulmonary artery is generally small and hypotensive. The left auricle is often tremen-

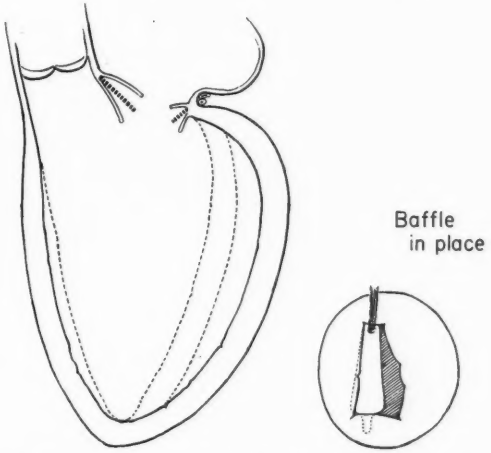

Fig. 6.—The bottle-baffle lay in a more favorable position for obstruction of the regurgitant jet and preserved leaflet mobility. Note bulging left ventricular myocardium and change in orifice size if leaflets are flexible.

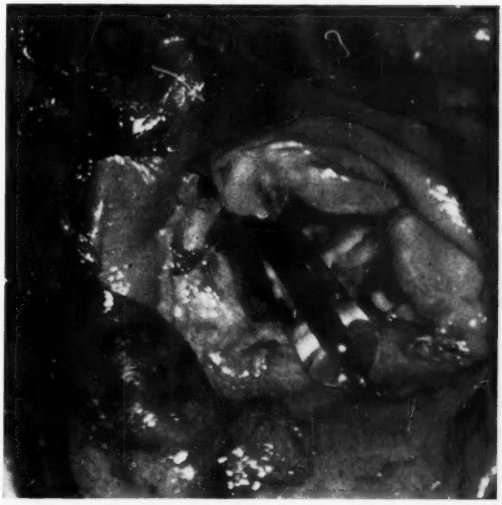

Fig. 7.—Autopsy specimen. The spindle-baffle is placed obliquely through the incompetent orifice and anchored firmly anterolaterally and posteromedially. The narrow anterior end is below, and the wider posterior end above, the annulus.

dous. The pericardium is opened posterior to the left phrenic nerve from above the pulmonary artery to the diaphragm in a line directed somewhat

dorsally. Retraction of the incised margins of the pericardium affords excellent exposure of the left auricular appendage, the lateral aspect of the left ventricle, and the phrenic surface of the left and right ventricles.

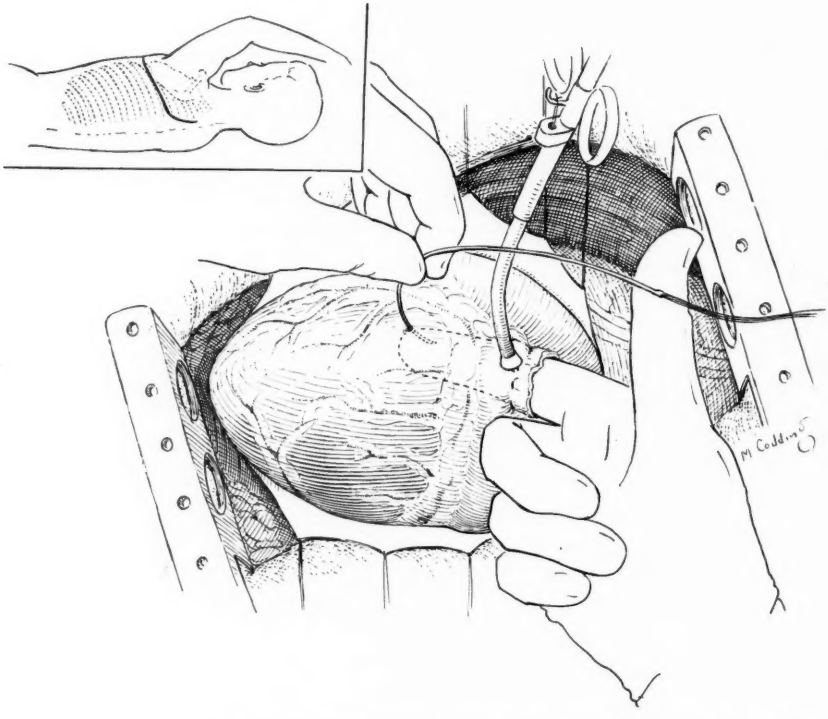

Fig. 8.—Technique of the spindle-baffle operation. Placing the anterior mooring of the baffle below the anterolateral margin of the annulus.

Purse-string sutures are then placed about the auricular appendage, the Rumel tourniquet is attached to one of these sutures, and the tip of the appendage is amputated with an auricle clamp controlling blood loss. The finger is then inserted into the auricle and the nature of the mitral incompetence explored. The finger is placed over the regurgitant jet in various positions and the patient's blood pressure is checked. The most effective position of the "finger-baffle" is that which produces the greatest rise in the systolic pressure as reported by the anesthesiologist.

The next step is to place a spindle-baffle of appropriate size and shape to simulate the salutary effect of the finger-baffle. As the surgeon palpates the orifice with the distal phalanx of his right index finger, he can compare various sizes and shapes of baffles with the corresponding phalanx of his left index finger thus making a reasonable selection of an appropriate prosthesis.

Almost invariably the greatest leak occurs posteromedially. The narrow end of the spindle is therefore placed anteriorly just beneath the annulus against the ventricular myocardium, and the broader extremity is brought up

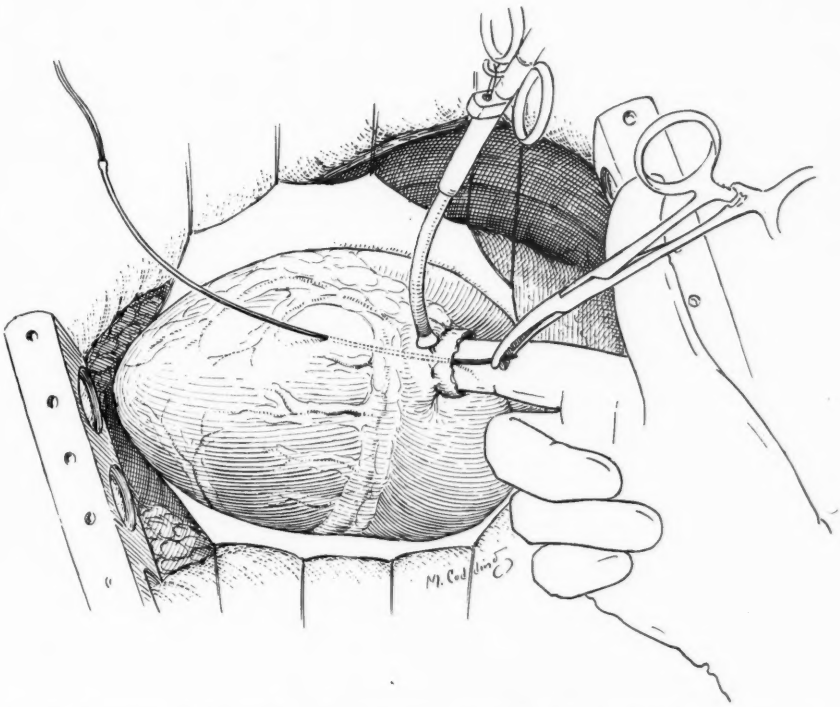

Fig. 9.—Technique of the spindle-baffle operation. Recovering the malleable needle from the auricular lumen.

through the orifice and fixed posteromedially just above the annulus. Thus, the prosthesis comes to lie obliquely through the mitral valve. It presents a smooth, nonreactive, polished Lucite surface to the leaflets as they close in systole and, because it does not interfere with their mobility, allows unhampered opening in diastole. The oblique position traversing the orifice ensures supplementation of leaflet deficiency regardless of changes in size of the annulus and/or the ventricle (Fig. 14).

The placing of the baffle is achieved as follows. A 12 cm. malleable needle with three strands of braided No. 1 nylon suture swedged on its end is tunneled through the myocardium so that it enters the chamber of the left ventricle near the anterior commissure (Fig. 8). The point of entry is in an avascular area between the anterior descending and the circumflex branches of the left coronary artery. It is then threaded up through the mitral orifice and out the appendage on the guiding finger as illustrated (Fig. 9). The

needle is passed through the appropriate spindle-baffle. It is then reinserted into the left auricle using the finger as a guide and directed so as to pierce the auricular surface of the posterior segment of the mitral annulus (Fig. 10). The needle is finally pushed through the ventricular myocardium beneath the coronary sinus and circumflex coronary artery, to exit from the heart just medial or just lateral to the posterior descending coronary artery on the phrenic aspect of the left ventricle. Thus, having in effect traversed the heart, the posterior end of the three nylon sutures is tied to a nylon button 1.5 cm. in diameter. By traction on the loop which is still emerging from the

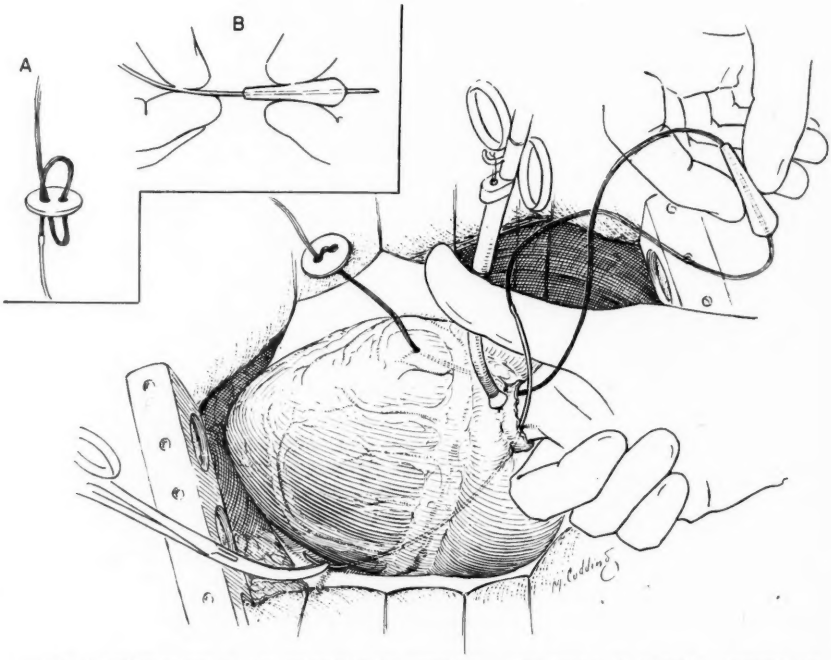

Fig. 10.—Technique of the spindle-baffle operation. The needle is threaded through the spindle from narrow to wide end. It is then reinserted into the auricle and through the auricular aspect of the posteromedial segment of the annulus.

left auricular appendage, the posterior button can be pulled firmly against the posterior wall of the left ventricle where it acts as the anchor for the broad posterior end of the spindle. Traction is next applied on the three nylon sutures as they issue from the anterior wall of the left ventricle drawing the spindle to the auricular appendage (Fig. 11). Further traction, properly timed with triangulation of the open end of the appendage and brief removal of the finger, makes it possible to "bob" the spindle into the heart without significant blood loss. As the anterior button is slipped along the three mooring sutures a completely stable position under any desired degree of tension can be attained (Fig. 12).

On numerous occasions the blood pressure has risen 30 to 50 mm. Hg immediately after placing the prosthesis, just as had occurred when the jet was obstructed with the finger. This observation would seem to substantiate the palpatory evidence of significant diminution in the volume of the regurgitant jet and indicate a salutary effect on the cardiovascular dynamics. The finger is then withdrawn from the appendage as the purse-string sutures are tied. Finally, the auricular margins are oversewn (Fig. 13), and the pericardium is loosely closed with a pad of Gelfoam over each button.

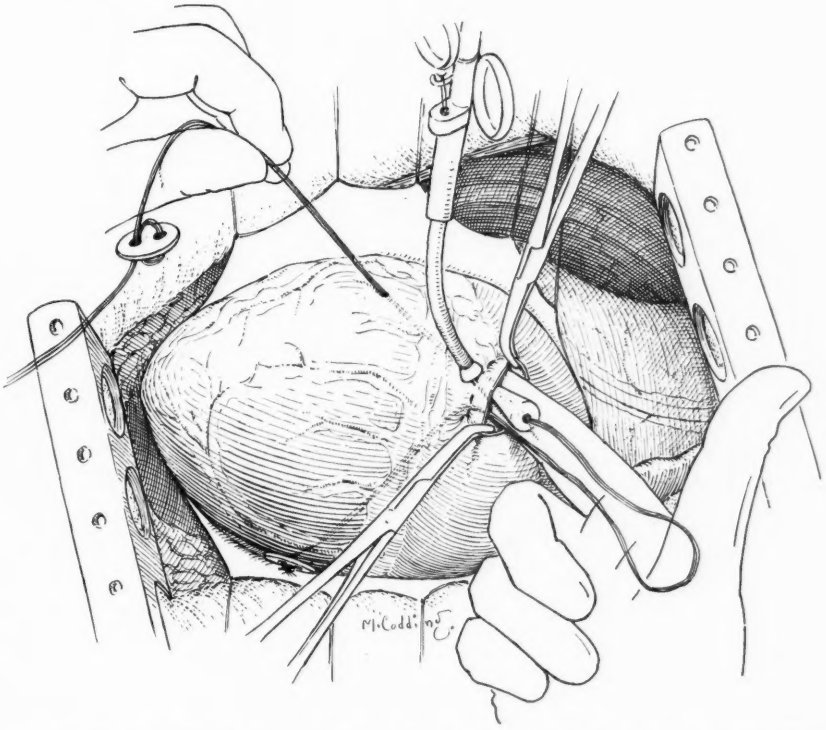

Fig. 11.—Technique of the spindle-baffle operation. Traction on the anterior (following ends) of the three No. 1 nylon sutures pulls the posterior button into place and draws the baffle to the auricle.

RESULTS

Among the seventeen patients who have had the spindle-baffle operation, there were three operative deaths.* The first loss was in a terminal patient who had been bedridden and unable to be brought into even a transient state of compensation. Baffling the leak with the finger resulted in no improvement

*One additional spindle-baffle operation was performed between completion of the manuscript and its presentation in Montreal. In this 16-year-old patient, the early postoperative death was felt to be related to active rheumatic carditis.

in the circulatory state at operation, and the blood pressure failed to rise. Presumably, the myocardium had failed irreversibly. Nevertheless, the prosthesis was placed simply as a last resort at this point. The patient awakened and responded following surgery but quietly expired within one-half hour with a progressively failing myocardium.

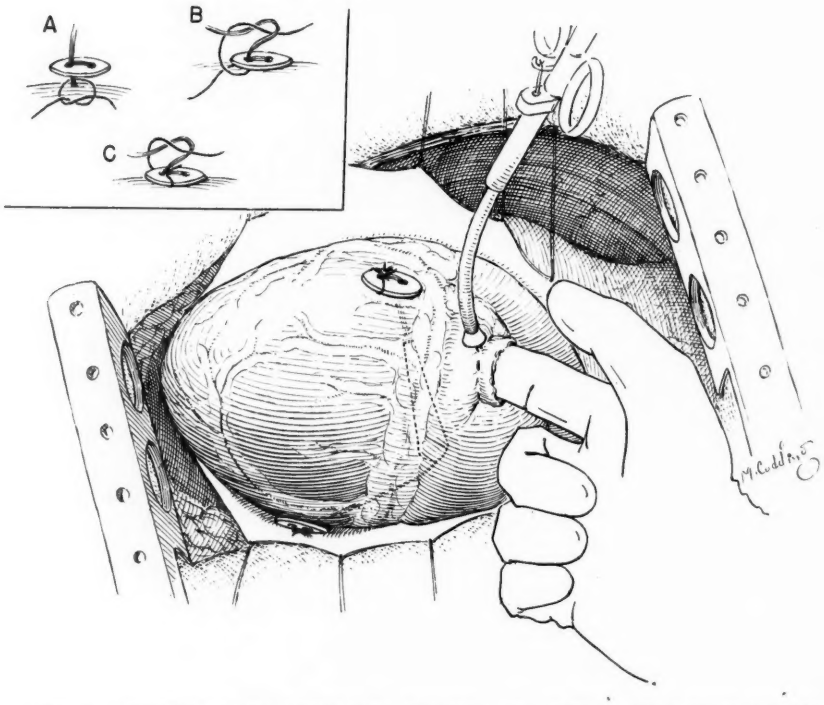

Fig. 12.—Technique of the spindle-baffle operation. The baffle is drawn firmly into the oblique position by traction on the sutures. The position is fixed by sliding the button down the sutures and securing it at the desired tension.

The second failure followed a technically simple operation, and the patient made the uneventful postoperative response similar to that we have come to expect after the valvuloplastic correction of mitral stenosis. However, after eating a hearty breakfast on the morning of the fifth postoperative day and making a phone call to her husband, she suddenly turned her head, gasped and expired within a few seconds. Autopsy revealed nothing more than a baffle in a completely satisfactory position (Fig. 7). The death is presumed to have been due to an acute arrhythmia similar to the Stokes-Adams attacks she had experienced preoperatively.

The third surgical death resulted from inadvertent dislodgment of a large clot from the auricle in a patient who had had a large femoral embolus re-

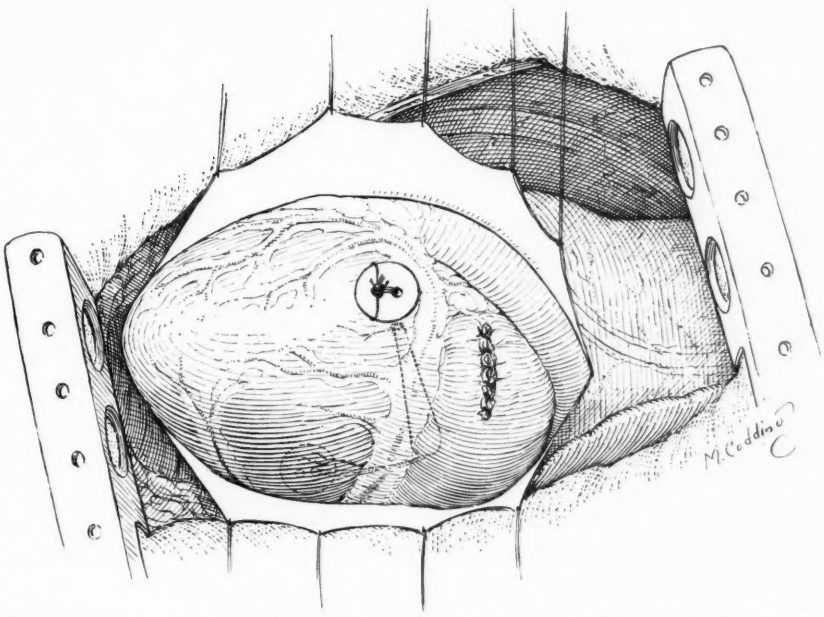

Fig. 13.—Technique of the spindle-baffle operation. The appendage is closed by tying the purse-string sutures and oversewing the margins of the auricle.

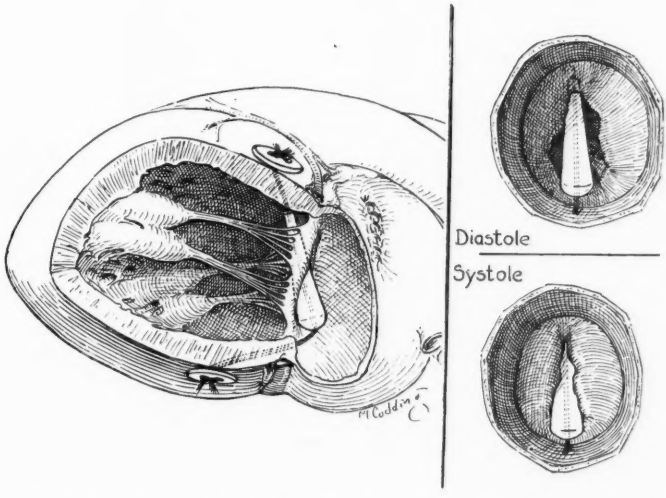

Fig. 14.—Technique of the spindle-baffle operation. Cut away detail of the position of the spindle-baffle. Note importance of leaflet mobility in allowing ventricular filling.

moved a few days before. This patient probably should not have been offered this procedure for the reason mentioned, and also because she had aortic stenosis and insufficiency. The one *late* death occurred in a man who was brought into partial compensation by prolonged medical treatment including one month in the hospital prior to surgery. The spindle-baffle further improved this compensation but one month after discharge irreversible congestive failure reappeared and led to his death.

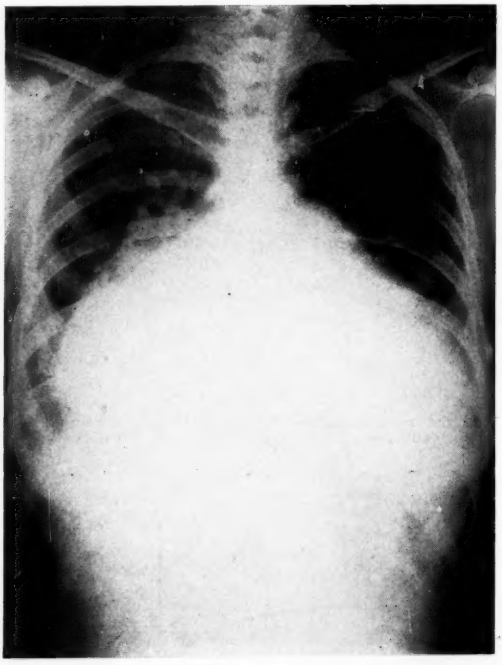

Fig. 15.—Roentgenogram of a patient in the spindle-baffle series. The left auricle comprises the bulk of the cardiac shadow extending to the left and right. Left ventricular enlargement is apparent in oblique views. Lung fields are relatively clear.

The surviving patients have had incomparably smoother postoperative courses than those patients who have had other types of surgery for the correction of mitral insufficiency. Furthermore, observation of the patients since discharge from the hospital is encouraging.

A conservative note must be sounded with respect to final evaluation of this procedure, because we have been disappointed before and the problem of mitral insufficiency is a much more complex one than that of mitral stenosis. The left ventricular myocardial component, at least in these terminal patients, represents a factor that makes the favorable response less dramatic, the result less immediate and less complete, and perhaps even renders good valve surgery less obvious. Certainly, if this procedure can help these terminal

patients and is associated with few or no late complications, it will afford more dramatic results when patients are accepted who do not have the crippling secondary changes (Fig. 15).

In the first year of our experience with the Lucite spindle-baffle operation for the correction of mitral insufficiency, it would appear that an operation reasonable in principle, simple of application, and salutary in effect, is available. The operation probably can be performed with a low surgical mortality comparable to valvuloplasty in the various stages of clinical illness.

The complexity of the clinical and pathologic problem, however, dictates that those patients whose clinical status is stable and who can safely await further evaluation of this procedure should do so. On the other hand, patients who have pure mitral insufficiency and whose condition is deteriorating on a good medical regime can conscientiously be offered this procedure.

SUMMARY

1. A morphologic classification of the pathology of mitral insufficiency is presented.

2. Some cardinal features of the kinetic pathology are described.

3. Various techniques that have been tried in other clinics to correct mitral insufficiency are reviewed.

4. Some of the problems incident to the surgical correction of mitral insufficiency are defined.

5. Various unsuccessful techniques in our experience are mentioned.

6. The rationale, technique, and results of the ball-baffle, bottle-baffle, and spindle-baffle operations are presented.

7. The spindle-baffle operation is considered sufficiently promising to deserve continued trial under restricted conditions in the human being.

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Discussion

(Papers by Bailey, Jamison, Bakst, Bolton, Nichols, and Gemeinhardt; and Harken, Black, Ellis, and Dexter)

DR. CHARLES B. RIPSTEIN, Brooklyn, N. Y.—The problem of mitral insufficiency is one with which Dr. Bailey, Dr. Harken, and many others have wrestled for a long time and, although it seems we are a bit nearer to a solution, there is still a long way to go. In analyzing the pathology in a good many cases we have seen at autopsy, certain facts have become quite striking to us.

In many cases the pathology seems to be a pulling down or adhesion of the mural leaflet in the posterolateral corner and with a flexible knife this can often be freed and divided and pulled up into place, in which the way Dr. Dodrill described about a year ago. We have found that this really does work and it is quite often possible to convert what appears to be an absolute lack of valve substance into a relative lack.

This is not always enough, because in addition to the shortening of the chordae and the adhesion of the mural leaflet the A-V ring is dilated and usually the posterior leaflet will not stay in position.

We have tried to hold it up by passing the suture under the leaflet in much the same way that Dr. Bailey's sling accidentally got there, but doing it with tendon, as Gordon Murray and others showed some time ago. Tendon grafts seem to be well tolerated in the heart and do not fibrose and shrink away. We have taken palmaris longus tendon and used that to support the leaflet and to fill the gap in the posteromedial corner.

In addition to that, we have experimentally used a purse-string suture to decrease the size of the annulus, but so far we have not had the courage to try this in the human being. To date, with freeing of the posterior leaflet, supporting it with a tendon graft, we have operated on 17 patients with mitral insufficiency, some of whom have had concomitant stenosis. There have been no deaths in this group and 14 patients are initially improved, but obviously the follow-up is still too short to be dogmatic about it. This is just offered as another way of doing the same thing.

DR. FOREST B. DODRILL, Detroit, Mich.—I should like to discuss primarily the type of deformity which I have encountered in seven consecutive patients in whom operation has been carried out for mitral regurgitation, and to illustrate a type of procedure which I have attempted in these patients.

The regurgitation is always at the posterior commissure. In six of the seven patients, it was the lateral posterior leaflet which was involved and approximately one-half of these had marked contraction and shortening of the chordae tendineae which held the posterior aspect of the leaflet down into the ventricle and prevented its closing during cardiac systole. The valve leaflet itself is not necessarily destroyed; in other words, if it is brought up into proper position so that it apposes the opposite leaflet, the regurgitation is minimized or corrected. In two of these patients the disease was definitely congenital, since one was a 3-year-old child and the other a 7-year-old child whom I had seen for a number of years. Most of my patients did not give a history of rheumatic fever and it is questionable whether all of them may not be congenital in origin.

The procedure which I carry out consists in inserting the finger into the atrium, passing it down through the valve to palpate the chordae tendineae and papillary muscle. If

this is found to be short and preventing valve closure, the finger is gently inserted under the valve leaflet and traction is made upward toward the atrium. By so doing, one gradually and gently stretches out the chordae tendineae until the blood, during cardiac systole, gets behind the valve leaflet and further tends to push it upward toward its proper position. That this mechanism actually occurs is borne out by the fact that in one of my patitents the murmur was still present after operation but after the course of two months almost totally disappeared. The first patient, upon whom I operated nearly two years ago, has been recently catheterized. It is seen that his pulmonary capillary pressure is now 8 mm. Hg, or essentially normal, whereas at the time of operation it was 30 mm. Hg. All patients have survived the procedure.

The point I should like to make is this: if there is sufficient valve leaflet for closure of the valve and if this valve tissue is simply distorted and pulled out of place, it is better to try to use this tissue and to put it back in the proper place if this is possible.

Mitral regurgitation is a severe disease and it is well known that a regurgitation of one-sixth to one-fifth of valve circumference decreases the cardiac output by at least 50 per cent, so that any degree of improvement is a great benefit to the patient.

DR. T. J. O'NEILL, Philadelphia, Pa.—At this point one thing should be quite clear: that there are a variety of types of mitral valve regurgitation and that no one specific type of technique or procedure will apply to all these situations. I should like to suggest one type of correction that has not been adequately explored at this time, that is, the use of living tissue rather than prosthesis and suture techniques, some of which will not be available at times.

Rather fortunately in some types of mitral regurgitation we find a large left atrial chamber and also a large left auricular appendage, and this tissue is available for use as grafting material. One can completely invaginate, in certain of these individuals, the left auricular appendage down through the mitral valve orifice and wrap it around the deficient posterior leaflet. This simulates Dr. Harken's finger-baffle maneuver: it places a bit of tissue that can be made without any special techniques into a tube which is approximately as large as the finger, and it can be anchored in a similar fashion as the prosthetic repair method through the annulus or below the coronary vessels.

Dr. Harken emphasized that the valve motility is important. Usually the mitral aortic leaflet is the large mobile saillike leaflet and it has adequate motion, as can be appreciated with the finger through the orifice, and this will move back and forth away from the bulk of tissue that is added to supply the deficient posterior leaflet, so that on diastole it will open, and the anterior leaflet will close against it in systole.

I will demonstrate its appearance in dogs. Here is the posterior leaflet, destroyed and replaced by the inverted auricular appendage. It is living tissue; actually it has no chance of dying, because only one or two sutures are placed through it. This is the aortic leaflet. It opens well. It is the only mobile leaflet. On systole it will close and wrap itself around that "door jamb."

I submit that certainly not all patients have situations in the valve that will lend themselves to this type of correction, but one does not have to worry much about what is going to happen to the bulk of tissue that is left in the heart. It is a part of the heart, it is living, there is no interference with its blood supply, and it will not move if it is anchored properly. We have operated on two patients with this technique without operative mortality. One is improved; the other, unfortunately, six weeks later lost the suture and there was recurrence of the regurgitation.

DR. EARLE B. KAY, Cleveland, Ohio.—As has been mentioned, the technique has to fit the condition found. I would like to discuss the group mentioned by Dr. Dodrill where the initial insufficiency results primarily from the valve cusp being rolled into and attached to the ventricular endocardial surface as a result of scarring and contraction of the rheumatic chordae tendineae.

In addition to attempting to elevate the valve edge digitally, the bound-down valve cusp can be more effectively mobilized by severing the more involved shortened chordae

tendineae and scar tissue by a small right-angle neurectomy knife which has been introduced through the left ventricular wall, using the finger within the mitral valve as a guide.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—As you know, Dr. Harken and I seldom see eye to eye on any new subject for several years, but eventually we do the same thing, even if we call it by different names. It is evident that we are becoming closer in our concepts and methods of management for mitral insufficiency.

This is the slide which Dr. Harken thought demonstrated a tricuspid valve. I do not think it is a tricuspid valve and they did not think so at the Institute of Human Anatomy at Copenhagen, from which I obtained the specimen. If Dr. Harken considers every wrinkle in the mural leaflet as an additional commissure he must deem most valves multicuspid. It took me six months to find a suitable human specimen of a mitral valve with an additional or commissural leaflet. Here is a picture of it. A quadricuspid valve is quite normal in the bull heart but Dr. Jesse Edwards of the Mayo Clinic estimates it to occur only in about 5 per cent of human cases. I do not know why Dr. Harken has found so many hearts of the bull type in Boston, but we do not find them in Philadelphia.

This slide is shown again to emphasize our old hammock or sling operation for mitral insufficiency and the spindle-baffle operation of Dr. Harken's. Although it is true that he allows his spindle-baffle actually to lie through the valve orifice, I can see that we are getting a lot closer together than we were.

I do object to Dr. Harken's repeated statements at medical meetings that our operation is designed to treat predominant stenosis and not regurgitation. Fifty-eight of our patients who were operated upon had no stenosis at all. And unless one wishes to exclude from treatment all patients who have some stenosis, it is necessary to treat many individuals with some stenosis because about three-fourths of patients with serious regurgitation present some narrowing of the valve orifice.

I see that he has had experience with 51 patients. Our operative experience has been with 150 patients, so I think we can recognize serious insufficiency and the results of surgical treatment at least as well as he can.

I have clearly indicated our early and late mortality on the statistical slide. Perhaps Dr. Harken will tell us the same about the 18 patients operated on by his latest technique. How many left the hospital alive, and how many are alive today?

DR. DWIGHT E. HARKEN, Boston, Mass. (Closing).—I shall have to send Dr. Bailey this manuscript. I said specifically, of the eighteen patients, four died in the hospital. That would mean that 14 left the hospital *alive.* One has died since, and unless someone has died since I was up here five minute ago, the others are living, and I might say doing quite well.

I do not wish to prolong this foolish exchange, particularly with reference to the discussion of bicuspid, tetracuspid, and tricuspid leaflets, but when the previous speaker showed his slide a second time, you may have noticed that there was quite a difference in the contour of the posterior commissural area and the anterior zone. You must either conclude that there was one continuous horseshoe leaflet or, as I contend, that there were two indentations posteriorly and, as is often the case, a separate set of chordae that clearly identify a posterior commissural leaflet. This is seen when the ring is open, and it is a common arrangement. Close scrutiny of specimens at the autopsy table will confirm this. Dr. Jesse Edwards has been quoted, and I must say that, except for a difference in percentages, we are in substantial agreement about the morphologic types. The colored slide showed an anterior commissural leaflet. Again, we are likely to see those things that we look for carefully; we cannot find those things which we do not know about. This exchange has reached the point of diminishing returns.

Dr. Ripstein, Dr. Kay, and Dr. Dodrill have presented an interesting and challenging technique. We shall give further attention to this maneuver. We have not found suitable types at autopsy or in the operating room in the past. Five years ago I performed five

operations by a similar technique and tried to stretch the chordae and leaflets. This was not very successful. No doubt time will clarify the situation, and we will arrive at a compromise. Further experience may even find proper places for such divergent concepts as are represented by the suturing of leaflets and the use of the inert baffle. The diversity of the pathologic types that we recognize admits of a difference of opinion between conscientious individuals.

Both left- and right-sided cardiac catheterization, which is an objective measure of hemodynamic improvement of fundamental importance in these early operations, may be helpful. Simultaneous pressure recordings across the mitral valve at the operating table may also be helpful. At this point our studies are inadequate. We can only say that the blood pressure does go up as the finger-baffle is placed; and if this finger-baffle is replaced perfectly by a Lucite spindle-baffle, the same increment of pressure takes place. We look forward to moment-to-moment cardiac output studies that will give the essential third dimension to this study. I have tried to present the spindle-baffle operation in the most conservative possible fashion. It just seems best to me to insert a nonreactive substance that does not change size or shape, *that does not move,* and that does not propagate thrombus, as does living tissue.

I have mentioned transbronchoscopic needle puncture of the left auricle with catheterization of the left auricle and, when possible, the left ventricle. We do not have adequate control of our catheter at the present time and cannot regard the technique as completely satisfactory. It does, however, seem to be reasonably safe, and we have carried it out twelve times without accident. More of these studies before and after surgery will be of great interest. The need for such an objective technique was made with reference to aortic stenosis. Dr. O'Neill has emphasized the importance of trying to remain objective, and we certainly agree with this. We look forward to giving you a subsequent report.

In conclusion, it is certain that we are dealing with a far more complex disease than mitral stenosis. The interrelationships between the myocardium, valve leaflets, valve orifice, lungs, liver, and so forth, constitute a problem that will require the most careful study by the most competent groups over a long period. We must not become enthusiastic about "corrective techniques" when incidental bed rest has brought about improvement or when we have infected the patient with our enthusiasm.

Erratum

In the November issue of the JOURNAL, the article entitled "A Neonatal Thoracotomy Retractor" by C. F. A. Cummins, Cambridge, England, appearing on page 546, was omitted from the Table of Contents. The publishers sincerely regret this error. An Erratum appears also at the end of the December Contents.

LIGATION OF CELLOPHANE-PROTECTED ARTERIES

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INTRODUCTION

IT OCCURRED to the writer that Cellophane may be of value in tying off fragile or sclerotic vessels, especially in cases of patent ductus arteriosus. Johnson and Kirby[1] emphasized that most patent ducti can be securely closed by ligation in continuity, and that recurrence is caused by failure to tie the ligatures tightly enough. They use size 0 silk and emphasize that umbilical tape or other heavy material binds upon itself, so that complete occlusion of the vessel is not at all certain. On the other hand, finer type ligatures can and have cut through the ductus. In older children and adults, the ductus wall may become friable and the pulmonary artery pressure elevated, increasing the risk of hemorrhage during surgery. Ligatures or clamps can cut through the vessel wall.

HISTORY

Since the original reports of ligation of a patent ductus arteriosus by Gross and Hubbard,[2, 3] Potts and associates,[4] and Touroff,[5] the trend has been toward complete division rather than ligation. Nevertheless, ligation is still widely used and is considered the simplest and safest procedure in many cases.

Holman and associates,[6] in a series of seventy-five patients with patent ductus, treated eight by division and sixty-six by multiple ligation in continuity. There was a recurrence in one case after ligation with one ligature, and the ductus was subsequently closed with multiple ligatures. In one patient the condition recurred after triple ligation, with formation of an aneurysm which was subsequently cured by division of the ductus at the site of aneurysm. One 14-year-old patient was found to have an aneurysm of the pulmonary artery opposite the entrance of the ductus. Bacterial endocarditis developed and was controlled by ligation of the ductus, wrapping of the aneurysm with Cellophane, and large doses of penicillin.

Starer[7] reported fifty-one cases of patent ductus arteriosus, all of which were ligated with heavy silk, without recurrence.

Possible Uses of Cellophane or Polythene in Ligation.—In simple in vitro experiments, use was made of ordinary thin "nonreactive" or nonirritating sheets of Cellophane, such as is found as a wrapper for packages of cigarettes and other common articles. Experiments were also made with thin sheets of

From the Veterans Administration Regional Office, Portland, Ore. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.
Photography by Mr. Parvin M. Eaton.
Received for publication April 9, 1954.
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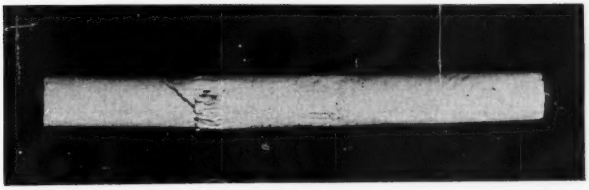

Fig. 1.—An ordinary cigarette which on the left has been clamped with a heavy Kocher clamp; note damage. On the right, three layers of thin Cellophane were first wrapped around the cigarette, before clamping; note that paper is wrinkled but cigarette is otherwise intact.

Fig. 2.—Left Figure: Left end of cigarette tied with 8-ply, twisted, heavy wrapping twine; it has cut through. Right end of cigarette, first protected with three turns or layers of a continuous strip of thin Cellophane, resists tearing or cutting through, even with more forceful tying. Right Figure: A "king size" cigarette has been first wrapped with three layers of a strip of thin Cellophane and then tied into a knot. After the knotted cigarette was untied, though wrinkled and distorted, it was free of tears and intact enough to be smoked.

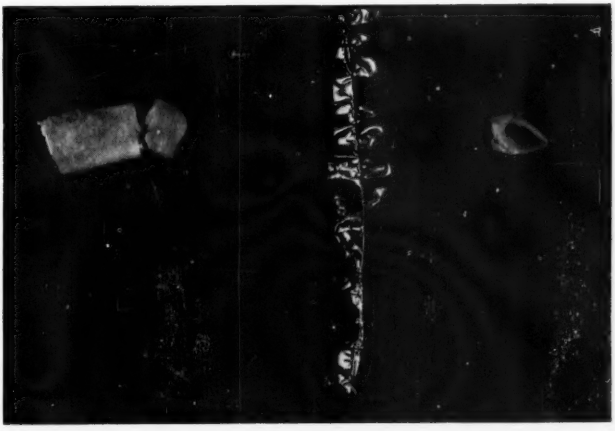

Fig. 3.—Left Figure: A sclerotic artery has been tied on its left end with extra strong "button and carpet" thread after first being wrapped with three turns of thin Cellophane; after removal of tie, the artery is grossly intact. The right end of the unprotected artery has been tied with the same kind of thread which is seen cutting through the vessel, even though less force was used in tying. Center Figure: The unrolled strip of Cellophane which was used to protect the artery shown in the left figure. The Cellophane is wrinkled, but grossly intact. Right Figure: Cross section of sclerotic artery used; it is 6 mm. in its outside diameter. The sclerotic left wall is 1.5 mm. thick, the less sclerotic right wall 0.5 mm. thick.

nonirritating polyethylene (Polythene), such as is ordinarily used to contain and protect poultry and vegetables in the household refrigerator. The protective value of Cellophane is illustrated in Figs. 1 to 3.

Wrapping an artery, such as the ductus arteriosus, with Cellophane or Polythene, and keeping the material closely applied to the vessel during its ligation, may be a somewhat difficult procedure when working within a rather small anatomic space. Fig. 4 shows a Cellophane strip prepared and ready to be managed as described in the legend. The width of the Cellophane strip would depend on the individual case. Care should be used to avoid injuring the recurrent laryngeal nerve with the wrapping material or ligatures.

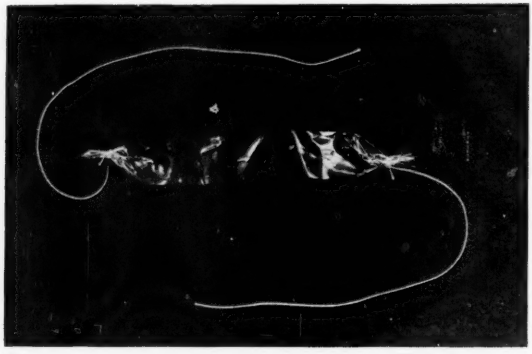

Fig. 4.—A Cellophane strip with a long piece of cotton thread tied at each end. One thread with attached Cellophane can be grasped with a fine Mixter-type hemostat and pulled under the ductus. This thread is now held taut with one hand while the other thread with attached Cellophane is pulled under, over, and around the vessel two or more times as desired, each time with the aid of a hemostat. Then, light traction on both threads keeps the Cellophane snugly applied to the artery so that one or more 0 silk ligatures can be applied to the Cellophane-covered artery. One central suture ligature can also be used, if deemed advisable, and if conditions are suitable.

It is seen (Figs. 1 to 3) that Cellophane is ruggedly resistant to tight ligature, yet is pliable enough to conform to, and mold itself down to, the object it is wrapped around. Although badly wrinkled, it shows no gross disruption of continuity along the line of the encircling ligature.

Polythene film is similar to Cellophane in its protective action, but to a lesser degree. It is softer and more pliable than cellophane and may therefore have its field of usefulness. But when wrapped around an object, it tends to "spring out" and unroll. It is less tenacious and protective than Cellophane, and ligatures tend to thin it out and cut through. It would also appear to offer less assurance, than does Cellophane, of preventing possible postoperative aneurysmal formation because of its more yielding nature, but may be less mechanically irritating to surrounding structures because of its softer texture.

DISCUSSION

The purpose of nonirritating (noncreative) Cellophane or Polythene film protection described previously is different from that described in previous

reports where, for example, Cellophane (cellulose hydrate or acetate) or polyethylene plastic film was used to wrap around arterial aneurysms, as described by Poppe.[8] It appears that early samples of polyethylene contained an irritating contaminant, dicetyl phosphate, which stimulated connective tissue formation; it was not the Polythene per se which was responsible for reinforcing the aneurysm. Since 1945 Polythene has been supplied free from this contaminant. When these facts became recognized, the irritating properties were deliberately utilized for connective tissue stimulation and reinforcement of aneurysms; a thin layer of dicetyl phosphate powder was melted and allowed to coat over the Polythene, the latter acting as a medium to hold the chemical.

It is interesting to note that Donald and Kirklin,[9] in experimental production of pulmonic stenosis, used one or two laminated Cellophane bands, 6.5 mm. in width, which were placed around the main pulmonary artery of dogs. In some instances, this produced tears in the pulmonary artery. If more than one or two bands were used, they combined to form a hard unyielding ring that ultimately eroded through the wall of the pulmonary artery. Again it is pointed out that the purpose and method of use of Polythene or Cellophane, as described in the present article, are entirely different from that of Poppe[8] or Donald and Kirklin.[9] My suggestion is that thin films of nonirritating (nonreactive) Cellophane or Polythene, wrapped twice or three times around an artery, protects it considerably against the trauma of ligature or clamping. Incidentally, these substances may act mechanically to discourage postoperative aneurysm formation.

SUMMARY

Nonirritating (nonreactive) Cellophane or Polythene film is suggested as an arterial wrapping before ligature, to prevent trauma, rupture, or postoperative aneurysm. Its possible usefulness for management of patent ductus arteriosus is discussed.

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COMPLETE TRANSPOSITION OF THE AORTA AND THE PULMONARY ARTERY

An Experimental Study of the Surgical Possibilities for Its Treatment

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COMPLETE transposition of the aorta and the pulmonary artery is a comparatively common form of congenital cyanotic heart disease. Abbot found in an autopsy series of 1,000 cases of congenital heart disease 5 per cent of complete transposition as compared to 8 per cent of cases with tetralogy of Fallot. These children are nearly always born in a good condition but have a very short survival time. After one month one-fourth of the infants had died and after one year more than three-fourths were dead. The surgical treatment must therefore be instituted during the first months of life.

In cases with transposition of the great vessels the left ventricular output through the pulmonary artery returns to the left atrium and the blood leaving the right ventricle through the aorta returns to the right auricle. Survival of these patients depends on some type of communication between the two circulations. It has been found in cases of complete transposition that an interventricular septal defect is associated with the longest life expectancy. An interatrial septal defect is the next most favorable isolated defect, and a combination of these two gives the best prognosis of all.

Blalock and Hanlon in 1948 devised an ingenious method for the production of an interatrial septal defect under direct vision without interruption of the circulation. They tried different shunt operations on patients with complete transposition of tne aorta and the pulmonary artery. The best results were obtained among the patients in whom an interauricular defect was created together with an anastomosis between the subclavian and pulmonary arteries. The patients were, however, only moderately improved and the arterial oxygen saturation generally remained below 75 per cent.

In 1953 Lillehei and Varco reported ten cases of venous transplantation in patients with complete transposition. Two patients survived the anastomosis of the right pulmonary veins to the right atrium and were improved. In four patients the inferior vena cava was shifted to the left atrium at the site of the cardiac end of the transected right pulmonary veins, the lung ends of which were anastomosed to the right atrium. None of these patients survived, a fact which was ascribed to an unbalancing of the circulation during the few minutes transpiring between the two anastomoses.

A direct switch-over anastomosis of the pulmonary artery and aorta with the aid of a "heart-lung" apparatus has been tried by Mustard without success.

From King Gustav V Research Institute, The Caroline Hospital. Head: Nanna Svartz, M.D., Professor; and Sabbatsberg Hospital, Surgeon in Chief: C. Crafoord, M.D., Professor.
Received for publication April 26, 1954.

In 1954 Bailey and co-workers tried the switch-over anastomosis of aorta and the pulmonary artery under hypothermia without survival. They considered this surgical approach basically unsound, since the coronary arteries nearly always arise from the right ventricular vessel.

Working with the aid of an extracorporeal circulation Gibbon has suggested that the coronary arteries have to be anastomosed as well.

ANIMAL EXPERIMENTS

As we have not had the opportunity to operate on patients with complete transposition of the great vessels, a series of animal experiments has been performed. A normal dog is less likely to tolerate any kind of cross transplantation than a patient with complete transposition.

These experiments were undertaken in order to work out a technique of switch-over anastomosis without interruption of the circulation.

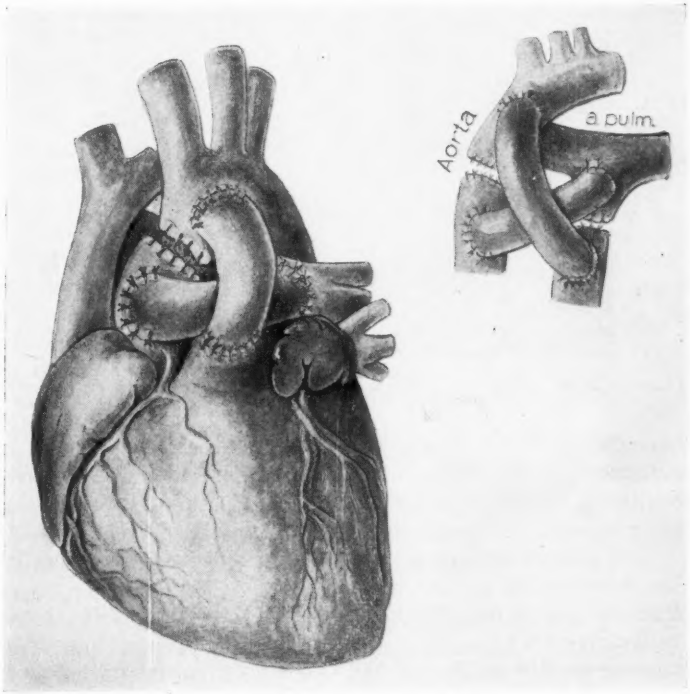

Fig. 1.—Drawing of a switch-over anastomosis with grafts between the aorta and the pulmonary artery.

In order to avoid the hazards of an extracorporeal circulation or hypothermia, the following technique was employed:

Dogs of 10 to 20 kilograms were anesthetized with Nembutal. A subcostal incision under the left fifth rib was performed. The pulmonary artery was freed to its origin from the right ventricle. The aorta was dissected free to

the origin of the coronary vessels. Then the clamp designed by Beck was applied over the anterior part of the aorta as close to the coronary arteries as possible. A longitudinal side opening was made in the aorta. Then an aortic graft kept in Tyrode's solution at –4° for a maximum of two weeks was sutured into the side opening. The posterior row of the anastomosis was performed with a continuous over-and-over five-0 silk on an Atraumatic needle

Fig. 2.—Photograph of a specimen from a dog with cross-transplanted grafts between the aorta and the pulmonary artery.

as recommended by Potts. The anterior row was best performed by isolated everting sutures. A continuous anterior suture on the very short part of the aortic wall inside the clamp had a tendency to cut through the brittle aortic wall. These holes were difficult to repair. The isolated everting sutures, on the other hand, can be used on a very short lip of brittle and unelastic aortic wall on old dogs without cutting through.

2. A similar anastomosis was made between the other end of the graft and the left pulmonary artery.

3. Another graft was then sutured to the main pulmonary artery as close to the right ventricle as possible.

4. The other end of the second graft was then sutured into the aorta distal to the left subclavian artery.

5. When both grafts were sutured in place, it was possible to place two Potts' ductus clamps for division on the aorta and the pulmonary artery between the anastomoses to complete the switch-over anastomoses. The clamps were only applied for a short time to prove the possibility of division and suture of the two ends. The clamps had to be applied obliquely to obtain

longer ends for suturing. These clamps were immediately removed and the wound sutured. The dogs survived with both grafts open for one to five hours only, when they died with pulmonary edema.

DISCUSSION

These experiments have demonstrated the feasibility of performing a switch-over anastomosis with grafts without interruption of the circulation in cases of complete transposition. All dogs died, as was expected, with pulmonary edema.

These findings will make the following conclusions obvious:

When cases with complete transposition of the great vessels are considered for surgery they must be divided in the following groups:

1. *Cases With High Pressure in the Pulmonary Artery.*—Approximately half of the cases of complete transposition are combined with an interventricular septal defect, 54 cases of 123 collected by Hanlon and Blalock; 14 cases of 29 collected by Lillehei and Varco had an interventricular septal defect as well. These cases with an interventricular septal defect and approximately the same pressure in the pulmonary artery and the aorta may be treated with a switch-over anastomosis with grafts obtained from babies who died at birth. The coronary vessels will still arise from the right ventricular vessel, but after a successful cross-anastomosis the oxygen tension of the venous blood would increase considerably.

II. *Cases With a Normal Pressure in the Pulmonary Artery.*—In cases with a normal pressure in the pulmonary artery a switch-over anastomosis cannot be used. Such an anastomosis would only result in circulatory arrest due to the low pressure in the left ventricle taking over the systemic circulation, or a pulmonary edema after a few hours due to the high pressure in the right ventricle taking over the pulmonary circulation as in the dog experiments.

In cases with a normal pressure in the pulmonary artery the only treatment possible is, then, venous anastomosis.

SUMMARY

Cases with complete transposition of the great vessels having a high pressure in the pulmonary artery may be considered for cross-anastomosis.

A technique of switch-over anastomosis with grafts without interruption of the circulation has been worked out on dogs.

Cases with complete transposition and a low pressure in the pulmonary artery cannot be considered for cross-transplantation. Venous transplantation is then the only treatment.

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TUBERCULOUS INFECTIONS COMPLICATING SUBCOSTAL PLOMBAGE WITH LUCITE SPHERES FOR THE COLLAPSE THERAPY OF PULMONARY TUBERCULOSIS

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PLASTIC ball plombage thoracoplasty introduced several years ago by Lucas and Cleland,[1] and Woods, Walker, and Schmidt[2] has found acceptance among many thoracic surgeons for understandable practical and theoretical reasons. It is an operation which offers relatively good selective collapse usually without multiple stages, freedom from postoperative paradoxical thoracic motion, and little or no interference with efficient cough and the expectoration of secretions. Deformity is considerably lessened. In addition, it has been shown by Watson and Gaensler[3] that the total pulmonary function sacrificed following this plombage procedure is less than after a standard thoracoplasty of similar extent, and that the return to the final postoperative functional level is more prompt.

The presence of a foreign body for protracted periods of time in the subcostal space has caused us some concern regarding the possibility of tuberculous infection from extrapleural tuberculous foci, or from the intrapleural disease by way of the lymphatics. Because of this we were led to the bacteriologic study of the fluid found in the subcostal space at the time of the operation for removal of the Lucite spheres and the rib resections performed to complete the second stage of this form of thoracoplasty. It is the results of these studies which form the basis of this communication.

During the eighteen-month period from July 1, 1952, to Dec. 31, 1953, thirty-two second-stage plombage thoracoplasty procedures, or "conversions," were carried out. The operations were all performed at approximately four months following the placement of the Lucite spheres. In our hands this interval, following the original operation, is optimal before the performance of the second stage. The free fluid in the subcostal space of each of these patients was examined bacteriologically by both immediate smear and culture for tubercle bacilli. Of the 32 cases converted, 12 were found to have positive cultures for tuberculosis from the fluid, and, of these, 9 had acid-fast bacilli demonstrable by smear at the time of operation. One of this group of patients who had a positive smear and culture returned eight months after conversion with a subscapular abscess and osteomyelitis of the eighth rib. Examination of the abscess material revealed the presence of tubercle bacilli.

Lucas and Cleland[1] reported one tuberculous space infection in 125 cases six months after placement of the spheres; Meyer, De Rougemont, and Labeur[4]

Received for publication June 7, 1954.

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found one late tuberculous infection in 20 patients at the end of four months. Wada[6] states that approximately 60 per cent of subcostal spaces in which the Lucite spheres were placed became infected with tuberculosis in Japan. The use of Lucite spheres in the extrapleural space was complicated by tuberculous infection in 21.5 per cent of the cases reported by Trent, Moody, and Hiatt.[5] In none of the above cases was there preoperative antibiotic preparation, and we concede that this was, in all likelihood, an important factor. All of our cases, however, had long periods of streptomycin treatment prior to surgery.

The postoperative accumulation of serosanguineous fluid in the subcostal space during the first two weeks following the placement of this plomb suggested to us that although the Lucite spheres were theoretically not reactive, this was not necessarily completely true.

This fluid accumulation was quite variable in amount, and in some patients daily tapping was required for as long as ten days after operation. Woods and associates[2] also recognized this problem and examined all the aspirated fluids from their first 45 cases, finding tubercle bacilli on only one occasion. These early postoperative studies on the infection rate in the subcostal spaces containing the Lucite spheres have probably led to a false sense of security. In our last ten patients study of this fluid revealed positive smears in one instance and positive culture in two other cases.

Since the presence of the subcostal Lucite sphere plomb led to a tuberculous infection rate in more than one-third of our patients, this procedure should not be recommended without reservation. We are cognizant of the increasing practice, on the part of some clinics, of leaving the Lucite spheres in the subcostal space permanently. The advisability of doing this, if it can be avoided, requires re-examination.

In view of these findings we feel unwilling to permit Lucite spheres to remain in a long period of time until accumulated experience shows this to be a safe course to follow.

COMMENT

Originally it was thought that the Lucite spheres would be less likely to be associated with late tuberculous infection of the collapse space than plombage materials such as air, fluids, or gauze used previously. On the basis of results of our bacteriologic studies, we are unwilling to be more than guarded when estimating the long-term outlook for these patients.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The Thirty-fifth Annual Meeting of the American Association for Thoracic Surgery will be held April 24, 25, and 26, 1955, in Atlantic City, New Jersey. Headquarters will be at the Chalfonte-Haddon Hall.

Requests for Hotel Reservations

These should be mailed directly to Miss Doris Sears, Convention Manager, Chalfonte-Haddon Hall, Atlantic City, New Jersey. Please mention this Association, the type of accommodation desired, date and approximate hour of arrival. If accommodations are desired elsewhere in Atlantic City, please communicate directly with the hotel of your choice.

Thoracic Surgical Forum

By vote of the membership, the Forum is to be continued for the presentation of short papers on current thoracic research, experimental problems, anatomical studies and modified or new surgical techniques. The inclusion of any considerable amount of clinical material in these reports makes the choice more difficult and the Program Committee reserves the right to reject such papers or to suggest their transferral to the regular program. Papers at this session will be limited sharply to ten minutes, including the showing of lantern slides or other illustrative material. At the discretion of the President, three minutes may be allowed as the maximum discussion for each paper.

These papers will be published together in one issue in the JOURNAL OF THORACIC SURGERY. Maximum length: 3,000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts for Papers

Abstracts for the presentation of papers at the 1955 meeting, including papers before the Forum, must be received on or before Dec. 15, 1954; otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Thoracic Surgical Forum," or "For Regular Program."

Five, repeat, five copies of each abstract must be sent to the Secretary of the Association, Dr. Paul C. Samson, 3959 Happy Valley Road, Lafayette, California. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words and accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee not later than Dec. 15, 1954; otherwise, the applications will be deferred for consideration until 1956.

Applicants must be sponsored by three Active or Senior members of the Association. In addition to the signatures on the application form, the sponsors must forward a *separate* letter concerning the applicant directly to Dr. Lyman A. Brewer, III, Chairman of the Membership Committee, 2010 Wilshire Blvd., Los Angeles 5, Calif.

Completed application forms and the sponsoring letters *independently* sent by the three sponsors must be received by Dr. Brewer not later than Dec. 15, 1954, to be considered for action in Atlantic City.

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Membership Committee should their applicants be held over for reconsideration a second year.

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